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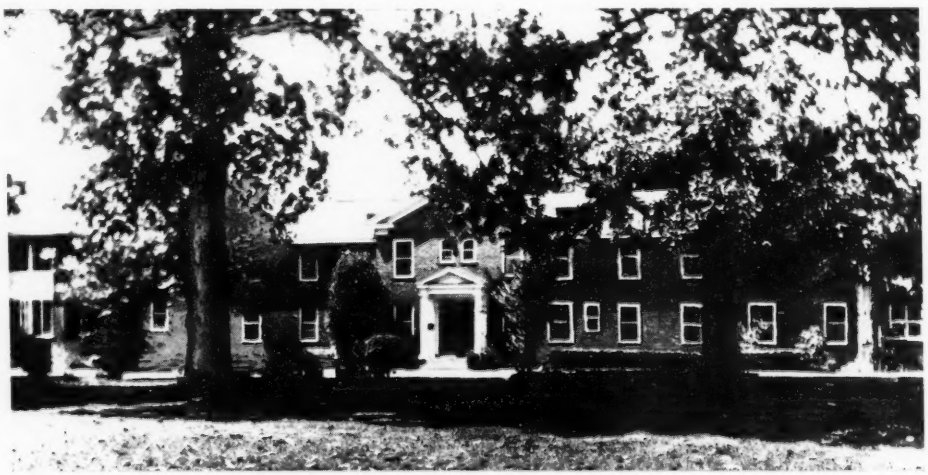

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Vol. XLIV **September, 1948** **No. 5**

DEVOTED TO THE INTERESTS OF THE MEDICAL PROFESSION AND PUBLIC HEALTH IN TEXAS

The State Medical Association Central Offices are now in operation at 700 Guadalupe Street, Austin, in accordance with a resolution passed by the House of Delegates of the Association last April.

The building occupied by the Association headquarters is a four story mansion built about sixty-five years ago by Mr. John Bremond, son of the founder of the John Bremond Coffee Company. For a number of years the house was used by the Elks Club of Austin, and during World War II the Austin Red Cross was located there. On a sloping lot at the corner of Seventh and Guadalupe Streets, three blocks west of the Stephen F. Austin Hotel, the building is of heavy stone construction with the external iron grillwork and interior mahogany woodwork which characterize many of the fine mansions of an earlier day. The building is not fireproof and of course was not planned for an office and library, but the steel desks and filing cabinets and the rows of bookshelves and journal stacks have fitted into the house fairly satisfactorily and the central office staff is prepared to go ahead with its usual routine.

State Medical Association Headquarters
700 Guadalupe Street, Austin, Texas

Actual moving from 1404 West El Paso Street, Fort Worth, the location which had served as Association headquarters since 1935, was accomplished mainly during the week of August 15, when van after van of office furniture, equipment, and supplies rolled between Fort Worth and Austin and household effects of the staff were transported from Fort Worth homes to the houses and apartments secured in Austin. Almost the entire personnel of the Fort Worth office decided to stay with the organization, only four of the full time employees deciding to remain in Fort Worth. While the actual move required a comparatively short period,

packing and unpacking, rearranging of equipment and supplies, and necessary readjustments consumed much more time, and the usual activities of the office were somewhat delayed in many instances.

The offices of the administrative assistant, with the public relations department, relocation service, medical economics division, and other divisions classified under the heading of the Medical Service Bureau, and office machines (addressing machine, mimeograph, postage machine, and so forth) are on the ground floor. The offices of the Secretary-Editor, the accounting division, the editorial and advertising departments of the JOURNAL, and the main files of the Association are housed on the first floor. The Library offices and reading room, book and journal stacks, reprint files, and motion picture films and equipment occupy the second floor. The third floor is being devoted primarily to storage of older books, back files of the JOURNAL, and other materials.

With the approval of the Trustees, the printing of the JOURNAL will continue indefinitely in the hands of the Stafford-Lowdon Company, Fort Worth, which has produced the publication since 1920 and is well acquainted with the requirements of the Association. All editorial, advertising, and circulation correspondence will be handled in Austin, however. Equipment for the annual session likewise will remain temporarily in Fort Worth under the supervision of Mr. L. E. Graber, who for many years has handled the technical exhibit booths and other physical equipment for the meetings; storage space for the equipment would have to be rented in Austin as the property at 700 Guadalupe Street has insufficient room for it.

The present quarters of the Association have been leased for three years, and arrangements are being made for the sale of the Fort Worth property. It is anticipated that within the next few years, the Association will have its own "made to order" building in Austin on land already donated by the Travis County Medical Society. The lot on Lamar Boulevard at the corner where Nineteenth Street will be cut through is an attractive site which promises sufficient room for a suitable building and for parking. Responsibility for drawing up building plans and for working out financial problems involved in the project was placed in the hands of the Trustees of the Association by the House of Delegates.

The central office staff invites the physicians of Texas to visit their new headquarters in Austin and to make use of the many services and facilities which are available there.

The Southwest Regional Cancer Conference will be held at the Blackstone Hotel, Fort Worth, on October 12. Sponsored by the Tarrant County Unit of the Texas Division of the American Cancer Society with the cooperation of the Tarrant County Medical Society, the conference is being held for the second year to provide scientific study for physicians of Texas and the surrounding states and to offer education in this important disease for laymen of the area.

Drs. A. R. Curreri, University of Wisconsin, Madison; Charles Huggins, University of Chicago; James Barrett Brown, St. Louis; Robert A. Moore, Washington University School of Medicine, St. Louis; and Morris K. Barrett, National Institute of Health, Bethesda, Md., will be presented in morning and afternoon sessions and an open forum luncheon for physicians and in an evening session open to the public. The public addresses will be especially designed to appeal to the interest and understanding of laymen.

The first annual Southwest Regional Cancer Conference received favorable comment from many quarters. The 1948 conference is already attracting interested inquiries from physicians and has been designated by the Texas Division, American Cancer Society, as one of its scientific conferences for the year. Among the subjects to be discussed are the place of the general practitioner in early diagnosis and management of terminal cancer; exfoliative cytology; chemotherapy of cancer; the solution of the cancer problem; and discussions of various forms of cancer including cancer of the face, mouth, stomach, prostate, and testis.

No registration fee will be collected for participation in the cancer conference, and physicians of the region are cordially invited by the committee in charge to take advantage of this one day program. Cancer is a subject of tremendous importance to physicians and laymen alike, and such an opportunity for receiving information at first hand from distinguished clinicians and teachers in the field should not be overlooked.

The Second North Texas-Southern Oklahoma Fall Clinical Conference will be sponsored by the Wichita County Medical Society in Wichita Falls on October 20 with four distinguished speakers from out of the state. Drs. W. Wayne Babcock, Philadelphia, surgery; E. R. Hayes, University of Minnesota, Minneapolis, medicine; L. A. Calkins, University of Kansas, Kansas City, obstetrics; and Hardy A. Kemp, Wayne Uni-

versity, Detroit, tropical medicine, will be featured in the one-day meeting.

Plans are being made for morning and afternoon lecture sessions, a noon luncheon, and evening dinner, all of which activities will be covered by a registration fee of $6. The conference will be held at the Wichita Falls Country Club. Visiting ladies will be entertained in a special program provided by the Woman's Auxiliary to the Wichita County Medical Society.

Interested physicians may secure additional information about the conference from Dr. Charles H. Brown, Chairman, 316 Hamilton Building, Wichita Falls.

The Wichita County Medical Society by providing annual postgraduate clinical conferences conducted by outstanding authorities is performing a real service to physicians of north Texas and southern Oklahoma who wish to keep abreast of late developments in medicine but who find it difficult to leave their practices to attend courses in centers at greater distances. It is to be hoped that members of the profession in the area will continue to support the venture which made such a good beginning last year.

Intern Placement.*—The House of Delegates of the American Medical Association in its meeting at Chicago in June of this year took action on a great many problems facing the medical profession of America. Among these was the intern problem which has been under study by a special committee appointed for this purpose.

The plan for intern placement adopted by the House of Delegates upon recommendation of this special committee, headed by Dr. William Bates, Philadelphia, consisted of specific rules for the use of senior medical students, medical school officials, and hospitals throughout the nation. These were as follows: Senior medical students may apply for internships only through the office of the dean of their school. These applications and credentials may be released to the hospitals on October 15 for internships beginning July 1 of the following year. The hospital administration may not commit the hospital or obligate the applicant before November 15 and no appointments may be made before that date. Applicants must accept or reject hospital appointments before midnight of November 18.

In connection with the intern and resident problem, the committee recommended in the interest of promoting general practice and of better preparing physicians to choose a specialty, that all specialty boards require at least one year of approved rotating internship before allowing a physician to start his specialty training. It was pointed out that an approved rotating internship should be of one or two years' duration and should include training in medicine, both organic and functional, obstetrics, and surgery, with special emphasis on diagnosis, under the supervision of a qualified educational director of interns. It was suggested that more internships be for a term of two years and that specialty boards should be requested to give credit toward certification for work in their respective fields during the second year.

The House agreed that hospitals at present approved for internships should be reappraised on the basis of their educational program for interns and that hospitals which do not provide adequate educational training programs for interns may be approved for general residencies for graduates who have completed an approved rotating internship of at least one year.

The Council on Medical Education and Hospitals of the A.M.A. was instructed to begin immediate implementation of the recommendations of the committee and to investigate and take appropriate action against hospitals and medical schools which do not cooperate with the plan of uniform intern placement.

This seems to be a step forward in making the distribution of interns in the various hospitals approved for internships more equitable and should go a long way in improving the training of interns in this country.

Individual Membership in the World Medical Association has been provided for physicians interested in promoting worldwide cooperation among the medical profession. A United States committee to aid the work of the World Medical Association has been established, and physicians of this country may become affiliated with the committee and become Founder Sustaining Members of the World Medical Association by payment of annual dues of $10, which also entitles them to receive the *Bulletin* of the Association.

The World Medical Association, described in some detail in the May, 1948, issue of the JOURNAL (page 8), has been endorsed by the House of Delegates of the American Medical Association, and various reputable professional groups within this country are organizational members of the Association. Dr. Louis H. Bauer, trustee of the A.M.A., is secretary-general of the Association. The national medical societies of twenty-six na-

*EDITOR'S NOTE: This is the first of a series of articles on the Transactions of the House of Delegates of the American Medical Association and activities of the various councils, committees, and bureaus of the A.M.A. which are destined to exert an influence on the practice of medicine and the improvement of the public health of this nation.

tions are already members, and sixteen others have applications pending.

Many of the great medical and pharmaceutical industries have expressed their approval of the World Medical Association by substantial contributions, but the Association cannot be established on a firm footing without the interest and cooperation of individual physicians around the world. As an editorial in *The Journal of the American Medical Association* (July 24, 1948, issue, page 1133) said:

> "The promotion of closer ties among the national medical organizations and the physicians of the world, the maintenance of the honor and dignity of the medical profession, mutual consideration of professional problems of the medical profession in the different countries, and exchange of information on matters of interest to the medical profession, the establishment of proper relations with similar groups (such as the World Health Organization, the United Nations Educational, Scientific, and Cultural Organization), improvement of health throughout the world, and the promotion of world peace are aims which merit support of every physician."

Physicians who wish to support the World Medical Association may write its permanent office at 2 East 103rd Street, New York City, for additional information or may simply send their dues for one or more years to that address.

CURRENT EDITORIAL COMMENT*

Treatment of Epilepsy—The first real attempt to direct treatment of epilepsy to the nervous system was made in 1857 by Sir Charles Laycock, who introduced bromides on the hypothesis that a sedative would decrease the number of attacks. In the early part of the twentieth century Hauptmann, following the same line of thought, substituted phenobarbital for bromides. The next major advance was made by Putnam and Merritt, who introduced dilantin sodium in 1937. Since then in rapid succession have come glutamic acid, tridione, and mesantoin.

With the advent of these new medicines there has been a revival of interest in treatment as well as renewed effort to find the basic cause of epilepsy. At present the outlook for epileptic patients is decidedly better; approximately one-half of those adequately treated with one or more of the anticonvulsant drugs can be completely free of attacks as long as the treatment is administered regularly every day. In an additional 25 per cent the seizures are greatly reduced in frequency. The remaining 25 per cent require special supervision in most of their activities.

The most common causes of failure in the treatment of epilepsy are not taking the prescribed dose of medicine regularly and inadequate doses of the anticonvulsive drug.

Frequently patients who have been well controlled and free of seizures for several months, with or without the consent of their physician begin reducing their dose or start skipping whole doses. This practice may result in a reappearance of their attacks.

Unfortunately it has been the custom to try to give the patient as little medicine as possible to control his attacks, leaving him with such small margin of safety that any unusual event such as an intercurrent febrile illness or a moderately severe hangover will induce a convulsion. The trend now is to give the patient maximum protection by increasing his medication until he just reaches the toxic level, then reducing the amount slightly so that he will be free of toxic symptoms. Of course this method requires careful observation on the part of the patient, family, and physician, but certainly the freedom from attacks makes this a worth-while procedure.

Another common cause of recurrence of seizures is changing abruptly from one medicine to another. If for any reason it is believed that the medication should be changed, this should be done gradually over a period of several months.

Finally, if the physician is to be successful in treating epilepsy, he must be willing to give more than just medicine. The utmost patience and understanding are required in dealing with the multitude of social, psychologic, and economic problems with which an epileptic person is faced. Epilepsy is apt to aggravate feelings of dependency and insecurity plus humiliation and anxieties fostered by public prejudice. It is often those nearest the patient who create most of his problems. Successful reeducation of relatives sometimes means the difference between success and failure in the treatment of an epileptic patient.

JOHN L. OTTO, M.D.,
Assistant Professor of Neuro-Psychiatry,
Director of the Epileptic Clinic,
University of Texas, Medical Branch,
Galveston, Texas.

*This department of the JOURNAL presents editorial comments on current items pertaining to the science, art and practice of medicine, contributed by members of the State Medical Association and scientists closely associated with the medical profession of Texas. Invitation is hereby extended to any member of the State Medical Association of Texas to submit such discussions for this department. The discussions should not be more than 500 words in length.

OBSTETRIC-GYNECOLOGIC PRIZE

The South Atlantic Association of Obstetricians and Gynecologists has announced the establishment of "The Foundation Prize." Authors of papers on obstetrical or gynecological subjects desiring to compete for the prize may obtain information from Dr. E. D. Colvin, Secretary-Treasurer, 1259 Clifton Road, N. E., Atlanta, Ga.

ORIGINAL ARTICLES

CLINICAL VALUE AND LIMITATIONS OF ELECTROENCEPHALOGRAPHY

MARTIN L. TOWLER, M. D.
GALVESTON, TEXAS

Like many of the other organs, the brain has more or less rhythmic variations in electrical potential. Electroencephalography is a means of recording and studying this activity and during the past ten or twelve years sufficient data have been accumulated to correlate variations in this activity with physiologic as well as pathologic changes in the brain. Without detailed consideration of the technique or theoretical aspects of electroencephalography, it is the purpose of this discussion to indicate the present clinical value and limitations of this diagnostic procedure.

The recording of the minute electrical potentials from the brain (roughly .01 that obtained from the heart with electrocardiography) is accomplished by the application of small electrodes to the scalp without pain or discomfort to the patient. Through a series of highly sensitive but powerful amplifiers this electrical activity is transmitted to a writing pen which makes a continuous record of the amplitude, frequency, and pattern of the electrical activity of the brain. It is generally believed that the activity recorded originates in the individual neuron but represents the synchronous and simultaneous discharge of a mass of cells. The postulation that the synchronization of this cellular activity results from a centrally located pace-making center[2] seems probable. It is significant to mention at this point that the activity recorded arises only from active or living neurons and that neurons completely destroyed by disease or injury no longer contribute to the rhythm. In an old or quiescent process, it is therefore possible to have a focus of destruction of neurons (without associated disturbance in the physiologic activity in the surrounding tissue) or rather extensive diffuse cortical destruction without significant disturbance in the electroencephalogram.

The criteria for a normal tracing have been reasonably well established by the study of a large number of presumably normal persons.[3] Like all other laboratory studies, there are considerable variations within the limits of normal and no clearly defined line should be drawn between normal and those with minimal abnormality. Furthermore, a small percentage of persons with known cerebral dysfunction will have normal tracings, and some presumably normal persons will have rhythms outside the limits of normal.

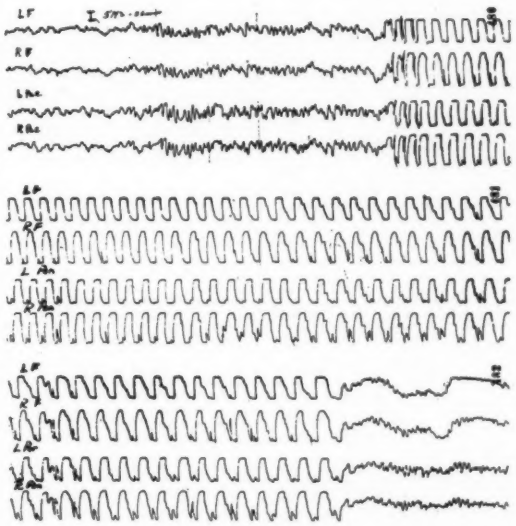

FIG. 2. Electroencephalographic changes in typical petit mal seizure of approximately 20 seconds duration. The figure is a continuous record of the seizure demonstrating the paroxysmal change of the electrical activity of the brain and typical spike and 3 per second slow waves. The patient was a white man, age 23, with onset of momentary "black-out spells" at the age of 20, later occurring at all times of the day on an average of two times daily. He had one grand mal seizure with tonic and clonic movements and loss of sphincter control. He was asymptomatic between attacks. Clinical diagnosis: petit mal and grand mal epilepsy.

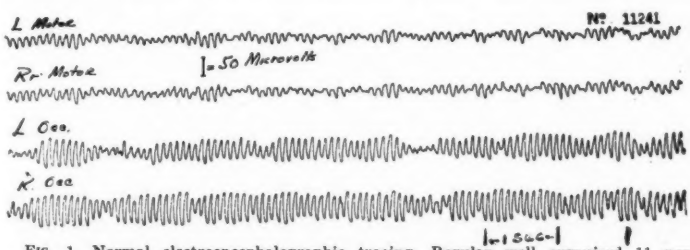

FIG. 1. Normal electroencephalographic tracing. Regular well organized 11 per second rhythm.

Similarly there are known physiologic and biochemical factors that produce alterations in the electrical activity of the brain, all of which should be known and taken into consideration when the tracing is interpreted. The most significant of these factors include age, degree of conscious awareness, factors known to alter cerebral metabolism such as blood sugar

From the Department of Neurology and Psychiatry, University of Texas School of Medicine.

Read before the Section on Medicine, State Medical Association of Texas, Annual Session, Dallas, May 6, 1947.

level, and drugs that stimulate or depress cerebral function. The normal adult pattern is usually established by the age of 13 or 14; prior to that age there is a variable but gradually decreasing amount of high voltage slow wave activity. Ideally, the most satisfactory tracings are obtained when the subject is relaxed with the eyes closed but not drowsy. Increased conscious awareness such as that associated with emotional tension, intense mental effort, or visual stimulation decreases the amplitude, regularity, and synchronization of the brain rhythm while decreased conscious awareness such as drowsiness or actual sleep causes the disappearance of the normal rhythm, which is replaced by irregular slow waves and later mixed fast and slow waves of increased voltage. Decreased blood sugar or oxygen tension decreases the frequency as well as the regularity of the pattern. Generally speaking, cerebral stimulants increase the frequency while depressants or sedatives decrease the frequency. Failure to recognize the significance of these factors may lead to erroneous interpretation.

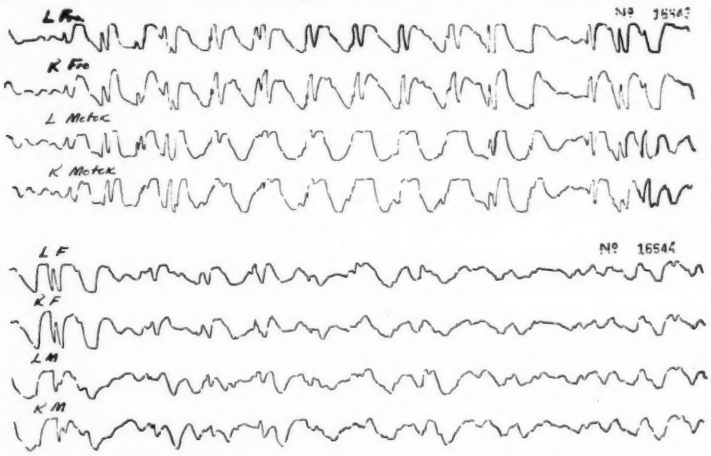

FIG. 3. Paroxysmal 2 to 3 per second slow wave activity with associated sharp wave and scattered spike components. The patient was a 17 month old infant with normal delivery, no serious illnesses, and normal development but with recent onset of fleeting spells of "staring off into space" during which time his attention could not be attracted. Clinical diagnosis: petit mal epilepsy.

CONVULSIVE DISORDERS

Since irregularly recurring disturbances in consciousness or conscious awareness with or without associated motor, sensory, and behavior phenomena may result from a large number of etiologic factors, it is extremely important from a therapeutic and prognostic standpoint to establish as clearly as possible the pathologic basis for such symptoms. Up to the present time the use of electroencephalography in such diagnostic problems has proved to be of more practical clinical value than in any of its other applications. In the majority of instances of persons suffering from various types of convulsive seizures, the history, observation of seizures and associated symptoms, course of the illness and ordinary clinical examination serve to establish the diagnosis and the electroencephalogram serves only as confirmatory data. In a significant number of such problems, however, the above data may leave considerable doubt not only with regard to the nature and degree of the disturbance in consciousness but also with regard to the cause for such symptoms. It is in this latter category that the electroencephalographic findings may be very helpful.

The characteristic finding in persons with known convulsive disorders is that of a sudden or paroxysmal change in frequency, voltage, and pattern formation (fig. 2 and 3). Less characteristic and less significant findings are those of frequencies that fall above or below the normal range of from 8 to 13 per second.

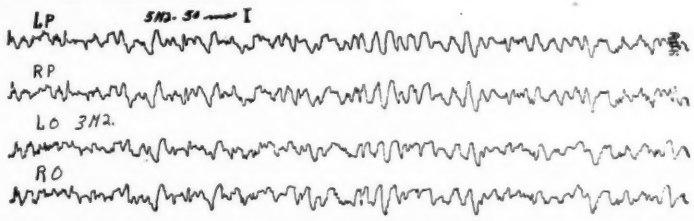

FIG. 4. Abnormally slow, diffusely irregular, and poorly organized activity with a great deal of 4 to 7 per second rhythm and relatively little normal activity. The patient was a 22 year old man with both grand mal and petit mal seizures since childhood.

In an extensive survey of 1,000 normal adult controls and 1,260 known epileptic patients of all ages Gibbs and others[3] have shown that abnormal tracings are found in 87 per cent of known epileptics as compared with 15.8 per cent of the normal controls. In the control group 87.3 per cent of the abnormal tracings represented minimal abnormality while only 29 per cent of the abnormal tracings in the epileptic group fell

into this category. The investigators found that paroxysmal dysrhythmias occurred in 38 per cent of the epileptic patients and in only 0.9 per cent of the controls. In other words, paroxysmal dysrhythmia is thirty-three times more common in epileptics than normal adults. Either very slow or very fast abnormal frequencies (fig. 4 and 5) were found in 20 per cent of the epileptics as compared with 1 per cent in the controls; moderately abnormal activity (fig. 6) in 29 per cent of the epileptics as compared with 13.8 per cent in the controls, and only 13 per cent normal in the epileptics as contrasted with 84.2 per cent normal in the control group.

These statistical data are informative but may be of little or no practical value in the individual cases. Obviously, the moderately abnormal activity, although it occurs slightly more than two times as frequently in convulsive states as in controls, may serve only to cloud the issue in the individual patient. Such is the case, however, with most laboratory procedures and in such instances repeated studies should be made. Experience has shown that patients with convulsive disorders may have reasonably normal tracings on one occasion, moderately abnormal activity on other occasions, and definitely abnormal paroxysmal dysrhythmia on still other occasions. Repeatedly normal tracings, however, would not serve to exclude positively the presence of a convulsive state. Not infrequently patients with hysterical syncope or fainting will state during the examination that they just had a "spell." The absence of coincident change in the brain rhythm at that time serves to eliminate the possibility of a convulsive state or syncope from other causes.

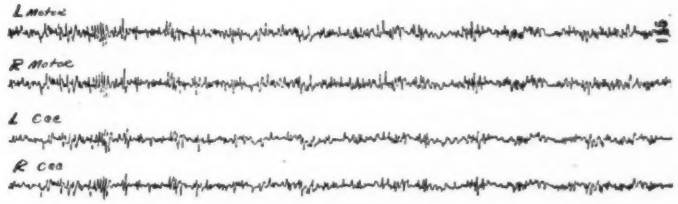

FIG. 5. Abnormally fast dysrhythmia. This tracing was made up almost entirely of 20 to 30 per second spike and fast wave activity and contained relatively little normal rhythm. The patient was a 33 year old man with grand mal epilepsy and "amnesic attacks" since childhood.

Patients whose histories include episodes of loss of consciousness, fleeting impairment of consciousness, transient alterations in behavior, or symptoms suggestive of Jacksonian seizures should be studied by electroencephalography unless the diagnosis is otherwise clearly established. In the majority of such instances the additional data obtained from the electroencephalogram are sufficient to clarify the diagnosis as far as the presence of cerebral dysfunction is concerned. In patients with a strongly suggestive history of a convulsive disorder, a single normal tracing should not be accepted as evidence to the contrary. Repeated studies should be made. The finding of bisynchronous paroxysmal dysrhythmia can be accepted as positive *laboratory* evidence of a convulsive disorder, very slow or fast activity as strongly suggestive *laboratory* evidence, and moderately slow or fast activity as questionable *laboratory* evidence of the presence of convulsive seizures. These data are then utilized in the light of other clinical and laboratory findings. The clinical diagnosis of a convulsive state is never arrived at on the basis of the electroencephalographic findings alone.

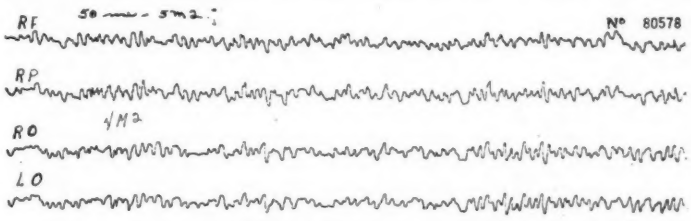

FIG. 6. Moderately abnormal tracing with an 11 per second dominant activity and scattered 5 to 7 per second slow wave activity in all leads. The patient, age 23, had had "dizzy spells" and "black-outs" since childhood; there was a positive family history for epilepsy. Clinical diagnosis: petit mal and grand mal epilepsy. In Gibbs series, tracings of this category were found to be present in 29 per cent of the known epileptics and 13.8 per cent of the normal control group. Repeated studies should be made in such cases.

Some effort has been made to use the electroencephalogram as an index to the effectiveness of anticonvulsant therapy. In my experience the electroencephalogram adds little if anything to the data obtained from clinical observation.

FOCAL EXPANDING INTRACRANIAL LESIONS

In the hands of a skilled technician, the electroencephalogram may aid significantly in the diagnosis and localization of focal expanding supratentorial lesions. Tumor tissue or that destroyed by an abscess is electrically inactive but the focal abnormality

results from disturbance in activity of the adjacent tissue.

Focal lesions usually give rise to a high voltage slow wave focus (fig. 7) and the degree of abnormality roughly parallels the acuteness or intensity of the disturbance created by the lesion in the surrounding tissue. Lesions accompanied by increased intracranial pressure or secondary vascular disturbances may give rise to diffuse changes as well as false localizing signs. Deep or centrally located lesions usually give rise to a diffuse disturbance in cortical activity.

When focal expanding lesions are suspected, the electroencephalographer should be so advised prior to the examination in order that additional electrodes may be applied. Unless electrodes are applied with sufficient proximity to the focal lesion, only normal activity may be recorded and the lesion overlooked.

When the hazards and discomfort associated with the two procedures are considered, the advantages of electroencephalography over pneumoencephalography are obvious. As yet, electroencephalography has not replaced pneumoencephalography but the persistence of a slow wave focus in keeping with clinical signs and symptoms can be accepted as strongly positive evidence of a focal lesion and may eliminate the need for pneumoencephalography or ventriculography. Little if any data can be obtained from the electroencephalogram with regard to the nature of the focal lesion, that is, neoplastic, inflammatory, traumatic, or vascular.

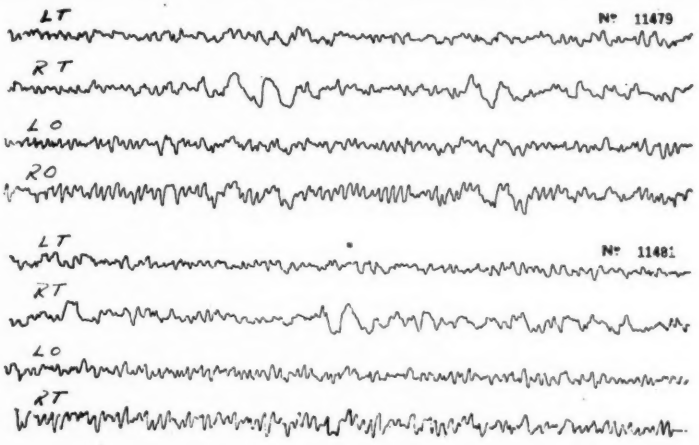

FIG. 7. Focal abnormality in the right temporal region without significant generalized disturbance. The patient had a pulmonary abscess but no neurologic signs or symptoms except bitemporal headache at the time of this study. Subsequent surgical exploration failed to reveal an abscess but at postmortem a large abscess was found in the right temporal region as well as multiple smaller abscesses elsewhere in the brain.

HEADACHES

Generally speaking, electroencephalographic studies on patients with headaches of undetermined origin add little to information obtained by other studies. Since most chronic headaches are either psychogenic or result from fleeting vascular changes, the tracings in the vast majority of such patients will be normal. The finding of minimal or even moderate diffuse abnormality in such instances may have no relationship to the clinical symptoms. The use of electroencephalography in psychoneurotic patients serves some useful purpose in that it satisfies their need for thorough physical investigation and affords them what amounts to a false sense of security. In rare instances patients with convulsive variants will present themselves with the complaint of periodic headaches and, without specific inquiry, no mention of the associated fleeting disturbances in consciousness may be made. In such cases, the finding of a paroxysmal dysrhythmia is helpful in establishing the diagnosis and points to the proper therapeutic approach. Engel, Ferris, and Romano[1] have shown that a slow wave focus develops in the occipital pole on the involved side during the prodromal stage of migraine. This finding is present only as long as the associated fleeting neurologic signs (scotoma) persist and is therefore of little clinical value. Electroencephalographic studies on patients with known migraine at other times reveals normal findings.

HEAD INJURIES

Although head injuries are accompanied by electroencephalographic changes, there has not been sufficient correlation between these changes and the patient's condition to be of any real clinical value. Information relative to the extent and reversibility of the damage is much more readily and accurately obtained by other clinical and laboratory studies. A record of electroencephalographic changes immediately following a head injury with subsequent reversal to normal may be of some medicolegal value but is of no clinical value. Persons with extensive traumatic brain damage, even to the extent of leading to a posttraumatic psychosis or deterioration, may have normal

tracings after the acute effects of the injury have subsided. There is no correlation between the common subjective complaints of posttraumatic encephalopathy and electroencephalographic changes except in those with posttraumatic convulsive states (fig. 8). It has been shown that focal abnormality is four times more common in posttraumatic epilepsy than in unselected epileptics.[4] This may be of some clinical value in that Jasper and Penfield[5] have shown that surgical excision of a clearly defined epileptogenic focus in carefully selected cases gives complete freedom from seizures or definite improvement in 71 per cent of the cases. I have had no experience with such a therapeutic approach; however, in view of the large number of focal penetrating head injuries incurred during the recent war and potential convulsive states resulting therefrom, electroencephalographic studies with such a procedure in mind are worthy of consideration.

The electroencephalographic changes in subdural hematoma are as variable as the clinical picture and may add little if any differential diagnostic data to the problem at hand. The findings may vary from a slight difference in voltage and pattern recorded from the two sides to that of a gross and diffuse abnormality in a comatose patient with increased intracranial pressure and secondary vascular changes.

MISCELLANEOUS CONDITIONS

Meningitis, encephalitis, vascular disturbances, and toxic reactions giving rise to delirium or disturbances in consciousness are all accompanied by changes in the electroencephalogram; however, these changes are nonspecific and do not add significantly to the clinical diagnosis. As the disease process subsides or becomes quiescent, the electroencephalogram usually resumes a normal pattern and is no index to the amount of residual damage. Occasionally the electroencephalogram may be helpful in differentiating acute toxic-organic reactions from acute psychotic disorders that give rise to stupor or mutism. In the latter group of disorders the electroencephalogram would be normal. During the acute phase of a cerebrovascular accident electroencephalographic changes are evident in the form of a focal abnormality or a diffuse irregularity depending upon the nature and extent of the lesion and the associated generalized disturbance. Usually within a few weeks a normal rhythm is reestablished even when there are evident residual neurologic signs. This fact may be helpful in differentiating vascular from focal expanding lesions in older persons. In the latter instance the focus would persist. The electroencephalographic changes in conditions such as hypertensive encephalopathy do not differ from those found in any condition giving rise to a generalized disturbance in cerebral activity including trauma, inflammatory reactions, or space occupying lesions with associated increase in intracranial pressure and vascular changes.

PSYCHIATRIC DISORDERS

In a study of delirious patients it has been shown[6] that there is a close correlation between the degree of electroencephalographic abnormality and the degree of disturbance in consciousness; however, there was no cor-

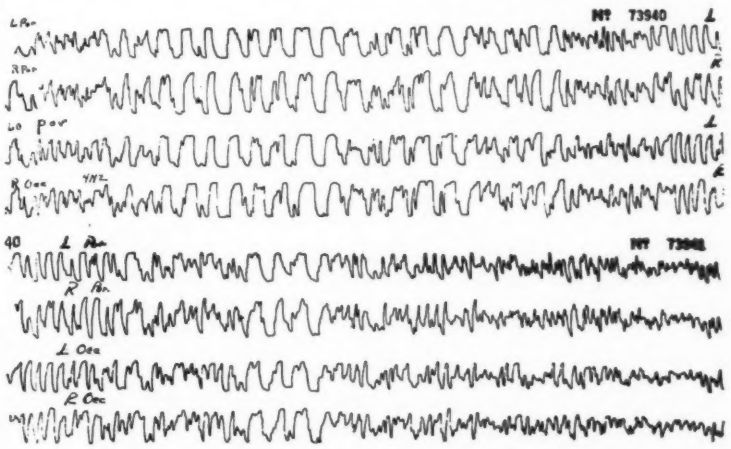

FIG. 8. Diffusely irregular and poorly organized activity with paroxysmal bursts of high voltage slow wave activity which is recorded simultaneously from both hemispheres. The patient was a 26 year old man who sustained a severe head injury with a twenty-four hour loss of consciousness at the age of 20 and started having grand mal seizures at the age of 22. He was otherwise asymptomatic at the time of this study. Clinical diagnosis: posttraumatic encephalopathy with grand mal seizures.

relation between the electroencephalographic changes and the patient's behavior or content of thought. Although a great deal of literature has accumulated relative to electroencephalographic changes in psychoneurotic disorders, psychopathic personalities, behavior disorders in children, and the various types of functional or psychogenic psychoses, there is no clear or conclusive evidence that disorders of this type are re-

lated to abnormal changes in the electrical activity of the brain.

SUMMARY

Electroencephalography is a diagnostic procedure that has all of the limitations common to any laboratory procedure and cannot be expected to replace sound clinical judgment. At the present time its most important clinical application has to do with the diagnosis or differential diagnosis of conditions that give rise to transitory disturbances in consciousness. In some instances of focal cerebral disorders, electroencephalography may replace pneumoencephalography or ventriculography. Inflammatory, vascular, traumatic, or degenerative disorders of the cerebrum are usually accompanied by changes in the electroencephalogram but these data have little if any practical clinical value. In only occasional instances, information obtained from electroencephalography is helpful in understanding the cause for chronic headaches. There is no clearly established correlation between abnormal changes in the electroencephalogram and psychogenic disorders.

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ABSTRACT OF DISCUSSION

Dr. William W. McKinney, Houston: Electroencephalography, as stated by Dr. Towler, is purely a laboratory test and is to be used as a diagnostic aid only. There are many factors which will cause variations in the normal tracings. An intelligent interpretation of these records can be made only in the light of an accurate history and clinical examination.

Electroencephalography is useful in diagnosing all types of tumors involving the cerebral cortex. Midline tumors and posterior fossae tumors tend to produce such generalized disturbances that no accurate localization can be made. Brain tumors in silent areas, such as the frontal lobe, may produce no abnormal neurologic signs. The presence of a tumor may be suspected on evidence so meager that it does not seem to warrant the discomfort involved in pneumoencephalography. Electroencephalographic tracings can be made without discomfort and at no risk to the patient and can be of considerable aid in diagnosis.

During the next few years, I believe that physicians will see a large number of posttraumatic convulsive disorders that have been produced by penetrating head injuries incurred during the recent war. Many of these cases can be benefitted by excision of the cortical scar. Fairly accurate localization of the epileptogenic focus can be made by electroencephalography.

SALICYLATE INTOXICATION

REPORT OF A CASE

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and
E. A. WILKERSON, M. D.
HOUSTON, TEXAS

Salicylates are probably the most common drugs prescribed at the present time. Ordinarily physicians regard these extremely valuable components of the therapeutic armamentarium as being almost without harm to their patients. Few realize that as little as 20 grains of acetylsalicylic acid taken orally have been demonstrated repeatedly to produce albuminuria in man and in experimental animals. In addition, urinary suppression, hematuria, and other signs of renal irritation are extremely common in instances where the ordinary therapeutic doses of salicylates are employed in the treatment of acute rheumatic fever.[7] In most cases these manifestations of salicylism in addition to nausea and epigastric distress are not of serious consequence. Hypersensitivity to salicylates, on the other hand, may produce drastic manifestations and may even become fatal. The total dosage required to produce these manifestations in the hypersensitive patient is extremely variable. One of the serious complications may result from a disturbance in the normal acid-base and electrolyte balances of the body. In such cases the initial manifestation of salicylate intoxication is usually hyperpnea of the "air-hunger" type, followed by fever, tinnitus, and mental confusion. Occasionally a toxic dermatitis consisting of a diffuse maculopapular eruption not unlike that seen in cases of bromide intoxication is noted. These symptoms may come on with alarming rapidity; consequently any delay in diagnosis is fraught with considerable danger. The following case report is given primarily to illustrate this point.

CASE REPORT

J. H., 21 year old Negro woman, was admitted to the Medical Service of the Hermann Hospital on June 30, 1946, with a typical attack of acute rheumatic fever. No previous episodes of a similar nature had ever been noted.

The physical examination at the time of admission revealed an acutely ill Negro woman lying quietly in bed. The oral temperature was 101 F., the pulse rate 125 per minute, and the respirations 22 per minute. The blood pressure was 130/80 in each arm. A few enlarged and tender submandibular lymph glands were palpable. The lungs were clear to percussion and auscultation. The cardiac borders were found to be within normal limits to percussion and neither ventricle was over-accessible. A soft systolic murmur was present at the apex. The pul-

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monary second sound was accentuated over the aortic second sound. The abdomen was essentially normal. No evidence of recent or old infection was demonstrable in the pelvis. The right knee, ankle, and wrist were found to be inflamed and acutely painful upon slight manipulation.

The leukocyte count was 16,900, and a heavy trace of albumin and from 8 to 10 white blood cells per high power field were observed in the urine. The sedimentation rate was 30 mm. at the end of one hour by the Cutler method. The Kline and Kolmer tests were negative. The electrocardiogram was within normal limits.

Following the patient's admission sodium salicylate in doses of 30 grains four times daily was prescribed orally and methyl salicylate was applied locally to the involved joints. The next morning, after a total of 60 grains of sodium salicylate had been administered, the patient complained of difficulty in breathing and ringing in both ears. Definite mental confusion appeared and the respirations became deep and regular. The superficial veins of the arms and neck became distended and the cardiac rate increased to 140 beats per minute. Tenderness was noted in the right upper quadrant of the abdomen, but the liver was not palpable and there was no evidence of pulmonary or peripheral edema. Early right sided cardiac failure was suspected and Lanatoside-C was administered orally in doses of 3 mg. immediately and 1 mg. every six hours for four doses. The mental confusion increased to the point of delirium by the following day, and the patient became dehydrated and appeared extremely toxic. The breathing continued regular, deep, and rapid. The possibility of salicylate intoxication was then considered and salicylate therapy was discontinued. Up to that time the patient had received a total of 120 grains of sodium salicylate during thirty-six hours. The carbon dioxide combining power of the blood was 33 volumes per cent and the blood salicylate level was 28.5 mg. per 100 cc. The body temperature, which had returned to normal the day following admission, rose to 102 F. Moist rales were noted in both lung bases posteriorly and a pleural friction rub was heard on the right. The liver enlarged to 2 cm. below the right costal margin and became tender.

Definite improvement was noted on the fourth hospital day, two days after salicylate therapy was discontinued. The gallop rhythm heard the day previous disappeared and the respiration approached normal depth and rate. A discrete maculopapular eruption was observed over the chest and both arms. These lesions varied in size from 1 to 4 mm. and contained a thin serous fluid. Cultures of this material showed no growth of bacteria. Several blood cultures were likewise negative. On the fourth day after cessation of salicylate therapy the patient was considerably improved and the carbon dioxide combining power of the blood was 46 volumes per cent. The breathing became normal on the sixth day post salicylate therapy and the sensorium was definitely clear. The following day therapy was again instituted in the form of acetylsalicylic acid, 10 grains two times daily with an equal amount of sodium bicarbonate. The joint manifestations, which had become aggravated during the interval in which therapy was withheld, promptly subsided the second day after treatment was resumed. The dose of acetylsalicylic acid was gradually increased to 60 grains daily without any untoward reactions. The pulse rate, sedimentation rate, and body temperature returned to normal and no evidence of cardiac damage was present on the nineteenth hospital day at the time of dismissal. The rash disappeared slowly but was completely gone at the time of the patient's discharge from the hospital.

She has been observed in the Out-Patient Department on several occasions following the above admission and signs of salicylate intoxication have not been present in spite of the fact that she has continued to take 60 grains of acetylsalicylic acid daily.

DISCUSSION

This patient had received only 60 grains of sodium salicylate at the time of the onset of her symptoms. The rapidity with which the symptoms progressed serves again to emphasize the importance of the early recognition of salicylate intoxication in order that serious and even fatal consequences may be avoided. This patient was critically ill for several days, but recovery was remarkably rapid following withdrawal of the offending agent. Whether or not the cardiac failure which preceded the signs of salicylism was precipitated by the reaction to the drug is problematical. Certainly digitalization did not produce the beneficial results that are expected ordinarily in cases of acute right and left sided cardiac decompensation. It was postulated, therefore, that the failure of the heart occurred because of the disturbances in the acid-base equilibrium, superimposed upon an already damaged myocardium. No evidence of cardiac failure was noted after the patient had recovered from the effects of salicylate intoxication.

Salicylate intoxication has become a subject of much interest in recent years, particularly since the advent of the use of massive doses both intravenously and orally in the therapy of acute rheumatic fever, as advocated by Coburn and his coworkers.[4] The mechanisms involved in the various manifestations of salicylism are poorly understood at present, despite much investigative work. True hypersensitivity to salicylate compounds occurs rather infrequently, as shown by Gardner and Blanton.[6] However, in a large series of cases studied at the Mayo Clinic Prickman and Buchstein[2] found that 10 per cent of all persons with an allergy of the respiratory system were sensitive to salicylates in one form or another. While these figures do not agree with other workers it is worth bearing in mind that people suffering from bronchial asthma may react violently to salicylates. Anaphylactic shock and exaggeration of the asthmatic symptoms are the serious reactions that may occur.

The total dose required to produce toxic manifestations is variable in different persons. Nausea, epigastric distress, vomiting, tinnitus, and mental confusion are the outstanding symptoms which precede the onset of the serious reactions. Gastro-intestinal symptoms are common during both oral and intravenous therapy. Recent studies on gastric secretions and gastroscopic examinations

during periods of intoxication have tended to disprove the idea that local gastric irritation is the basis for these symptoms. No salicylic acid or evidence of gastric irritation was found by Paul[11] and by Caravati and Cosgrove[3] in studying patients taking massive doses of salicylates. Hyperpnea is another common manifestation of salicylate intoxication. A direct stimulation of the respiratory center in the brain by the drug is thought to be the mechanism involved. The degree of hyperpnea is practically always out of proportion to the decrease in the carbon dioxide tension of the blood. Also, hyperventilation not infrequently persists for some time after the carbon dioxide combining power of the blood has been restored to normal levels. As a rule, hyperventilation does not occur until the blood salicylate level has reached 400 micrograms per 100 cc. of blood. The excessive loss of carbon dioxide through prolonged hyperventilation results in a decrease in the alkali reserve of the blood. It might be assumed that a period of compensatory apnea would follow the diminished carbon dioxide tension of the blood. However, the fact that this does not occur is further proof that the hyperpnea results from a direct stimulus to the brain by the drug.

The metabolic disturbances are by far the most important features of salicylism. The initial alkalosis resulting from the excessive loss of carbon dioxide is quickly followed by a ketosis according to some workers. Others are of the opinion that the picture is one of acidosis from the onset and that the salicylate effect upon the blood electrolyte pattern counteracts the effect of the hyperventilation. A rapid depletion of liver glycogen and an increase in the lactic acid content of the blood in salicylism has been demonstrated experimentally by Lutwak-Mann[10] and by Dodd and her coworkers.[5] These studies reveal that salicylates in "toxic" doses suppress tissue oxidation and certain enzymatic reactions in the cells of the liver. This results in an accumulation of lactic acid, sodium lactate, phosphates, and certain fixed acids in the blood. In an attempt to compensate for the decrease in concentration of the bicarbonate ions, the lactic acid is broken down to yield carbon dioxide. A decrease in the level of the serum sodium and potassium has been demonstrated by Rapoport and Guest.[8] This is due, probably, to the combination of these substances with the fixed acids in the blood and a consequent loss in the urine. Transient acetonuria occurs rather frequently and may persist for some time in cases of severe intoxication.

These same workers[13] stated that in cases of experimentally produced salicylism there is a lowering of carbon dioxide tension of the blood, a decrease in the serum sodium and potassium, and an elevation of the chloride content of the blood. They found that in the early stages the total electrolyte pattern of the blood is maintained within normal limits by the elevation of the serum chloride. In the later stages, however, the chloride ion is unable to compensate for the loss of the bicarbonate ion and acidosis ensues. These experiments also demonstrated that salicylates in toxic doses tend to augment the untoward or convulsive side reactions of barbiturates and opiates in animals.

Hypermetabolism, as evidenced by an increase in the basal metabolic rate, is a constant finding in salicylism. Dodd and her coworkers[5] have stressed this point in an attempt to explain the rapid depletion of liver glycogen. These workers have suggested a specific effect on carbohydrate metabolism by salicylates, resulting in incomplete oxidation and the formation of ketone bodies and other organic acids. Albuminuria and an increased urinary output are also common and have been considered by some to represent the results of a direct toxic effect on the kidneys by salicylates. Others believe that these findings are the result of excessive excretion of electrolytes, namely, sodium and potassium. Hematuria and granular casts are often noted in severe cases, in addition to albuminuria.

Occasionally a diffuse papular acne-form rash is noted in cases of salicylate intoxication.[1a] These lesions usually appear on the second or third day following the onset of symptoms and disappear promptly as the other manifestations subside. Recently cases of hemorrhagic encephalitis due to salicylate intoxication have been reported.[1] A specific effect upon the vascular system of the brain similar to that noted in instances of arsenical reactions is apparently the basis of this complication of salicylate therapy. There does not seem to be any correlation in these cases between the prothrombin concentration of the blood and the hemorrhagic manifestations in the brain. The effect of salicylates on the erythrocyte sedimentation rate and the prothrombin concentration of the blood is still unsettled. A rapid fall in the erythrocyte sedimentation rate with the onset of salicylate therapy has been noted on numerous occasions and is explained on the basis of a decrease in the level of the serum fibrinogen, which is thought to be directly related to the sedimentation rate of the red blood cells. However, others, notably Jager and Alvary,[9] have commented on a distinct rise in the

sedimentation rate in several of their cases of acute rheumatic fever with the onset of therapy. Quick[12] was the first to demonstrate the decrease in the prothrombin concentration of the blood during salicylate therapy.

SUMMARY

The clinical manifestations of salicylate intoxication are hyperpnea, tinnitus, mental confusion, and fever. At the onset there is an alkalosis of the carbon dioxide deficit type due to the extreme hyperventilation as a result of a direct stimulus to the respiratory center by salicylates. This is rapidly followed by a severe acidosis which if not recognized promptly may terminate in convulsions, coma, and death. These events may transpire within a period of from thirty-six to forty-eight hours in the patient who is hypersensitive to salicylate preparations. While this condition is not common, it is worth while to emphasize the importance of being aware of the potential hazards when these drugs are used.

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ABSTRACT OF DISCUSSION

Dr. Charles T. Stone, Galveston: This paper is timely and important because of the frequency with which salicylates are employed in treatment, particularly in rheumatic states, and because serious and at times fatal intoxication may result from their use. Salicylate intoxications have been notably much more frequent since the employment of massive salicylate therapy in rheumatic fever regardless of whether the drug is given orally or intravenously, but especially so when the intravenous route is used.

Most clinicians now agree that a high level of plasma salicylate, that is, between 35 and 40 mg. per 100 cc. of blood is most effective in controlling the features of acute rheumatic fever such as pain, fever, tachycardia, leukocytosis, and increased erythrocyte sedimentation rate. However, there is widespread disagreement with Coburn's advocacy of administering from 10 to 20 Gm. of sodium salicylate intravenously daily. Equally as high blood levels can be obtained by the oral administration of the drug with a marked lessening in the incidence of toxic reactions. Furthermore, many cases of acute rheumatic fever respond as favorably to from 5 to 7 Gm. of sodium salicylate daily as they do to the larger doses recommended by Coburn and others. Admittedly the exceptional case will not be controlled with the smaller doses. Patients vary greatly in their ability to concentrate the drug in the blood after both oral and intravenous administration. A recent contribution by Dry, Butt, and Scheifley has shown that unsatisfactory blood levels may be increased to desirable levels by the concomitant administration of 24 Gm. of para-aminobenzoic acid daily along with the salicylates.

With regard to the so-called acidosis that complicates salicylate therapy, and which was present in the case reported by Drs. Bernard and Wilkerson, the mechanism is apparently as follows: In susceptible persons a considerable concentration of salicylate in the blood acts as a stimulant to the respiratory center in the medulla. This causes hyperventilation with a consequent loss of carbon dioxide, and in this condition more carbonic acid is blown off through the lungs than Bicarbonate. This results in the finding of a diminished blood carbon dioxide but a rise in the hydrogen ion concentration in the blood. This state has been called a "respiratory alkalosis." Thus there arises the somewhat anomalous situation in which there is an increase in the alkalinity of the blood with a diminished blood carbon dioxide, which is the common clinical measure of acidosis.

In treating this condition, the obvious thing to do is to recognize it early and discontinue salicylate therapy promptly, at least temporarily. In those instances in which the hyperventilation continues, the best course is to employ a remedy which will inhibit the respiratory center and for that purpose morphine sulfate is the drug of choice.

In the case reported, the authors were fortunate in being able to continue salicylate therapy by substituting acetyl salicylic acid for sodium salicylate. They did not furnish figures on the salicylate level after acetylsalicylic acid treatment, but the obvious conclusion is that the level was lower than the level obtained when sodium salicylate was employed and that it was below the level of toxicity for that particular patient.

I would like to emphasize that the salicylates in rheumatic fever constitute largely empiric therapy. Their action is analgesic and antipyretic, and they have a tendency to limit the exudative features of the disease. They do not affect the incidence of carditis except perhaps indirectly by shortening the course of the acute manifestations of rheumatic fever. Since rheumatic fever affects predominantly mesenchymal structures, the principal substrate of which is hyaluronic acid, it is important to note that the salicylates have been proved to inhibit the action of hyaluronidase, which favors the diffusion of the toxic agent of rheumatic fever into the involved structures. An advance in the knowledge of these enzyme systems may have an important bearing in the future therapy not only in rheumatic fever but many other diseases caused by bacteria and perhaps by other ultramicroscopic agents.

ADVANCES IN INFANT NUTRITION

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One of the many bright spots in medical progress is the increased knowledge we now have of proper nutrition for infants and children. This paper will give a brief resume of some of the most outstanding and practical advances in nutritional knowledge that have been described in recent pediatric literature.

In the feeding of premature infants, it has long been believed that human milk was the most desirable food. The mother of the premature infant cannot often furnish breast milk and it is difficult or expensive to obtain it otherwise. But in a recent study[3] of 122 premature infants comparing the results obtained with cow's milk and human milk, it was found that partially skimmed cow's milk is superior, especially for the smaller premature infants. This is attributed to the greater protein and mineral content of cow's milk as compared to human milk. Since partially skimmed cow's milk is always available, this is a valuable aid in solving the feeding problems of premature infants.

Also in premature infants or in other infants who are not gaining well, it has been found[7] that the giving of human blood plasma orally is of great value and actually amounts to giving an "oral transfusion." The giving of plasma orally was suggested by the similarity of colostrum to plasma. If Nature provides this human protein solution for newborn infants, physicians also can give it orally in the form of plasma when indicated.

For acutely ill infants who can take no food orally, it is now possible to provide adequate nutrition intravenously. Not only may carbohydrates be given in the form of glucose solution and proteins in the form of amino acids, but fats have been given intravenously also. In 1944, a report[4] was made of 1 infant whose entire caloric needs were met by intravenous feeding for five days. The intravenous fat given consisted of a homogenized emulsion of olive oil and lecithin.

A practice that is becoming obsolete, I hope, is the routine prescribing of formulas for newborn infants. The loss of weight that occurs during the first few days of life is physiologic and is the result essentially of the loss of fluids in the urine, feces, and perspiration. When a formula is given to a newborn infant, he is not given a chance to become hungry. He does not nurse at the breast vigorously. Since vigorous nursing by a hungry infant is the greatest stimulus to breast milk production it is important not to remove this powerful and natural stimulus by satisfying the infant's appetite with a formula. Physicians can compensate for excessive weight loss by giving adequate fluids orally or subcutaneously, and prescribe formulas only in those unusual cases where the need actually exists.

A rigid feeding schedule is no longer advised. No clock knows when a baby is ready to nurse. The baby should be fed when he is hungry. He should be left alone when he is contented or asleep. The baby will soon develop his own schedule, which will be reasonably regular. This "self-demand" schedule works beautifully and is natural.

Neither is it advisable to prescribe a time limit for feeding. Some babies nurse rapidly and others slowly. The sucking instinct is more pronounced in some, and if they are not allowed to nurse until they are ready to stop, they are likely to develop the habits of thumb and finger sucking[11] as a compensatory mechanism.

MILKS

Of the various milks available, breast milk still ranks first. Every mother should be encouraged to nurse her baby. The mother who offers some trivial excuse for not wanting to nurse her baby may often be persuaded to nurse the baby "for only the first month." Then she can be counted on to continue breast feedings after the first month because she wants to.

Next to breast milk, cow's milk is the most valuable, while goat's milk has only a limited use in certain allergic infants. The anemia that is associated with the use of goat's milk may be prevented or cured by the use of iron and liver.[9] For the family that does not have a refrigerator or cannot be depended upon for careful preparation of the formula, lactic acid milk is the safest. Pathogenic bacteria will not multiply in lactic acid milk, and lactic acid milk does not require refrigeration.[13]

Several trends are apparent in the construction of cow's milk formulas. One trend consists in the inclusion in the milk of the necessary vitamins. The daily vitamin D requirement of an infant is from 400 to 800 international units, with twice that amount required by premature infants during the first year.[12] Furthermore, since rickets has been found in about 45 per cent of children up to the age of 15, it is now recommended that all children up to 15 years old receive their daily vitamin D requirements.[2] If the daily requirements of vitamin A and D are

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included in the milk in a readily assimilated form, the child will automatically get the vitamins even when the mother becomes forgetful. Vitamins A and D are included in many evaporated milks, while one brand has in addition vitamins B_1 and C plus iron and other minerals. There are many suitable multi-vitamin preparations that may be added to any formula. Vitamins should be started by the second month and given consistently.

Another trend in the prescribing of formulas is the reduction and early elimination of the carbohydrates added to the formula. Some have even advised never adding sugar in any form to the formula,[8] claiming that the children develop better, are not so fat, stand hot weather better, and are more active. The evidence submitted would support this view.

It has been demonstrated[6] that infants as young as 6 weeks have tolerated strained meat added to the formula. One ounce of strained meat added to each day's formula increases the protein intake by 25 per cent. This checks the usual drop in the hemoglobin level and promotes the formation of hemoglobin and red blood cells.

Still another trend that may help in preventing allergic rashes is the prescribing of ascorbic acid instead of orange juice during the early months of infancy. In early infancy, the intestinal tract is more permeable to various substances and may admit proteins that are allergenic. By giving ascorbic acid rather than orange juice, many cases of orange juice allergy may be avoided. For the same reason, precautions must be taken in the feeding of egg. Since egg white is more allergenic than egg yolk, only the yolk is permitted in early infancy. Later, toward the end of the first year, whole egg may be allowed.

SOLID FOODS

It is now customary to start solid foods by the second or third month. Most infants will receive fruit sauces well but many will reject cereals. Since the feeding process should always be pleasant for the baby and the mother, I give fruit sauce first, starting with small amounts and gradually increasing them. Another advantage of fruit sauce over complex foods such as cereal mixtures is that fruit sauce is a single substance rather than a mixture of many substances. This is important when an allergic rash makes it necessary to identify the offending factor. New foods should always be introduced one at a time and in small amounts which may be gradually increased. After a food has been given for five days in liberal amounts and no allergic rash has appeared, it is unlikely that the child will have any allergic difficulty with that food. He is then ready for the trial of one other new food, but only one at a time.

Sooner or later a food will be introduced that displeases the child and he will spit and gag. Such a food should be withdrawn promptly as no one food is worth a fight. If that same food is offered some weeks later, especially at a time when he is hungry, it may be eagerly accepted not only then but thereafter.

Solids should be given first at a mealtime before the baby gets his milk. After 6 months of age solids will play an increasingly important role and the milk consumption will be automatically reduced to a pint and a half or less per day. The addition of cream cheese to the diet at this time will prevent any deficit in calcium and phosphorus intake. The dairies of the country have sold most mothers on the idea that every infant and child should have at least one quart of milk daily. To counteract this propaganda, specific limitations must be prescribed on the milk intake; otherwise some children who love their milk will almost limit their diet to milk alone and will develop into anemic, flabby children.

It is important at the ages of 7 to 9 months to shift from the pureed vegetables and fruits to the coarser foods. If this change is not made early enough, many children with a sensitive gag reflex will resist the coarser foods effectively. The junior chopped foods are designed to meet this need. However, many children will accept table foods better than the junior chopped foods. To sit at the table and eat with the family is a delightful social experience for almost any child. When foods are mashed a little with a fork or spoon, they will swallow and digest almost anything within reason. They will eat more and better if they are allowed to feed themselves.[1]

Another well established principle that is useful in the feeding of the child of 1 year and older is the "self-selection" of foods.[1] Appropriate foods are offered in variety and the child is allowed to choose what he wishes to eat and to eat as much of any food as he desires. Although this results in unpredictable variations from day to day, the over-all food intake is invariably well balanced. The child thrives and enjoys his meals. Today, he may eat two bananas and three eggs, while tomorrow he may fill up on meat and cereals. Adults should allow for children as they do for themselves a variability of appetite for foods both as to selection and quantity.

When suitable foods are offered in variety, it is advised that a child be allowed to choose what he wants to eat and how much he wants to eat. This will result in a good diet, a happy child, and eating habits free from complexes.

"RUNABOUT" CHILDREN

Finally, I wish to consider the problem of the child of runabout age who has a poor appetite. This problem is made more acute if the child is an only child with an oversolicitous mother. Something can be done in the way of preventive therapy if the mother is told during the child's early infancy that his appetite will be much decreased after the first year; that this is a physiologic process since the child does not triple his weight each year as he has the first; and that his increase in height added to his slower gain in weight will make him appear slim or even skinny; that what food he does eat goes to stature and activity, rather than to weight. Since one quart of milk will supply at least half of his caloric needs, it is advisable to cut the milk intake to one pint daily, and even this pint may be partially skimmed.[5] Since it has been established that one glass of milk between meals does not impair appetite at mealtime,[14] this pint may be given between meals, leaving the stomach at mealtime free for the intake of solid muscle building foods.

There are several things that can be done to encourage a child to eat better. In addition to prescribing that vitamin B be given to the child for the mother's benefit, I suggest that she refrain from undesirable activities at mealtime such as persuading, bribing, coaxing, or forcing her child to eat. Only small portions are to be served the child and if the food is refused it is to be removed without comment or apparent disappointment. Most children prefer only two or three foods at a meal.[1] If the child is an only child, the mother could form a working agreement with other mothers of only children and let their children have meals together. Children always eat better in the company of other children, especially if the meal is served picnic style. Reasonable allowance should be made for a child's preferences for foods, and if he wants ice cream for breakfast, it may be served. If the child wants fried foods, these may be served since it has been established that properly fried foods are not harmful to children.[10] Fresh fruit given between meals actually increases the mealtime appetite.[1]

Nearly all children love hamburgers. If the ingredients are selected and prepared at home, a nutritious meal can be incorporated into a hamburger containing lean meat, sliced tomatoes, lettuce, mayonnaise, relish, shredded cabbage, and carrots. One juicy hamburger with a glass of milk is a wonderful meal. The protein foods such as lean meat, cheese, and eggs are extremely valuable for growing children and are usually well taken. With patience and forethought, the "runabout" child can be encouraged to eat well and enjoy it.

SUMMARY

1. Pediatric literature in recent years has been full of practical suggestions for the improvement of nutrition of infants and children.

2. By following these suggestions, physicians can make life happier for their little patients, their mothers, and themselves.

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ABSTRACT OF DISCUSSION

Dr. J. R. Lemmon, Amarillo: Since I am a clinician I shall confine my remarks to clinical application of nutrition. The knowledge and teaching of pediatric nutrition has improved remarkably since I was a student. However, there is still much to be attained in the teaching field. More stress should be given to nutritional deficiency symptoms and simple correction of faulty diets in infants and children. For example, during the past two years I have hospitalized several infants with definite clinical scurvy in which the physicians had completely missed the diagnosis and the mother finally in desperation consulted a pediatrician. The painful limbs had even been treated for rheumatic fever in one case and in no case had a diet history been taken. The physician had not thought of a nutritional deficiency but was thinking in terms of diseases.

The regular feeding patients are scarcely ever acutely ill in a well controlled pediatric practice. Hypochromic anemia is the most common condition found in nutritional deficiency cases. In my experience breast feeding has almost disappeared and the infant has suffered as a result. Pediatricians are often blamed for artificial feeding—but do we not

find that most infants have already been weaned from the breast when the mothers finally consult us as a last resort for poor nutrition? With the wealth of usable information at our command we should be better feeders and at least think of nutritional deficiencies and thus solve a lot of simple problems in every day practice.

Dr. Gibson mentioned the premature infant. Breast milk is still the first choice. Dryco is an old stand-by and is safe because of its lower fat content. A dangerous tendency in feeding a premature infant is to try to get too rapid a gain in weight. A premature infant should not be pushed too fast on caloric feeding and essential vitamins should be added early. A child should not be fed by the clock, as Dr. Gibson has stated. The old Army dictum of "come and get it and take it or leave it" is good to follow in anorexia and some nutritional problems.

The vitamin racket is not as bad as it was a decade ago. However, the detail man still presses physicians too much with samples and pseudoscientific data aimed chiefly at boosting sales. The young physician should carefully consider all claims made before adopting a vitamin product. Simplicity and adherence to basic rules of nutrition will save a lot of trouble and promote better development of young patients.

USE OF GAMMA GLOBULIN IN PEDIATRICS

CLINICAL OBSERVATION

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Clinical and experimental investigation in the use of immune serum for virus infections is remarkably limited and the results have been to a large extent disappointing. This unsatisfactory state is possibly due to the general difficulties attending virus research such as inability to grow the virus easily and abundantly so that hyperimmune serums could be produced, the low titer of viral antibodies in the serum following a natural infection, limited supplies of immune serum, and difficulty of early diagnosis of the natural disease. Despite these handicaps and despite the general concept that antiviral immune serums are valueless, it has been my belief that the study of the effect of immune serum should continue.

The evaluation of its worth can be finally ascertained for the most part only by tabulation of clinical results. The chief aim in this paper, therefore, is to keep open the consideration of the value of immune bodies in the prophylaxis and treatment of diseases known to be caused by virus, and other diseases in which the agent is still somewhat obscure. Another aim is to offer cases indicating that the belief that cells already parasitized by virus are beyond any help from serum therapy is not entirely accurate if a sufficiently potent source of immune bodies is used.

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What is immune serum globulin or, as it is perhaps better known, gamma globulin? Blood is composed of cells and plasma which can be readily separated by such simple physical means as centrifugation. About 6.5 per cent of the plasma is protein. It is this element which is chiefly responsible for the benefits obtained in plasma transfusions. The protein in plasma is a mixture, chiefly of albumin, fibrinogen, and alpha, beta, and gamma globulins. These proteins differ in molecular size and structure and in physical and therapeutic properties. The separation, concentration, and drying of the different components of plasma by the use of physiochemical methods was accomplished first by Edwin J. Cohn[2] at Harvard University in 1947.

Gamma globulin makes up about 11 per cent of the total proteins of plasma. Its therapeutic value is due to the fact that all antibodies against infectious diseases which are present in plasma appear to be concentrated in gamma globulin. Antibodies against disease are manufactured by the body not only if the disease is actually contracted but also following exposure to the disease; they remain in the blood stream for varying periods of time. It should be emphasized that the process of fractionation not only separates but also concentrates the antibodies. The concentration of antibodies in the gamma globulin, thus prepared, is approximately twenty-five times that of normal pooled plasma.

A brief reconstruction of the probable behavior of the virus in the susceptible host may throw more light on what may be expected from specific treatment. Viruses differ fundamentally from bacteria in that they are intracellular parasites. This property has long been thought to make this infectious agent immune to therapeutic measures. In practically all other respects, however, virus infection and immunity to virus infection are comparable to bacteria infections and immunity. As T. M. Rivers said, "There is nothing peculiar about immunity in virus diseases. Principles that hold in other fields operate also in the virus domain."[5] The infectious agent enters the body through one or more portals of entry and invades body cells. Multiplication of virus in these body cells takes place. The virus after a period of time escapes and is transmitted through one or more pathways to other cells of the body. The virus may invade adjacent cells, it may invade the blood and lymph stream, or it may extend along nerve fibers. The method of spreading probably varies considerably in different infections and may play an important

part in determining the efficacy of any specific therapy. Such continued spreading and multiplication must be progressive until the maximum extent of cellular invasion occurs. Therefore, there are certain salient facts which must be reviewed briefly in order that specific prophylaxis or treatments can be judiciously considered:

1. Virus infections are followed by a variable amount of antiviral substances in the blood stream. Although not demonstrated conclusively, it appears possible from the facts now available that either apparent or unapparent infection with the more common viruses in childhood or early adult life effects a slowly developing permanent immunity to such infection in the greater part of the general population.

2. For a serum to be effective it must be rich in viral antibodies. The pooling of plasma from hundreds or thousands of adult blood donors, while of only quantitative value in the separation of such plasma components as albumin, may be and usually is of great qualitative value in respect to gamma globulin.

3. Immune serum can neutralize extracellular virus in transit and at the site of parasitized cells in the body.

4. Immune serum surrounding susceptible cells will probably prevent infection of these cells by virus if it is present in sufficient potency.

5. In some diseases if the virus has invaded cells the presence of immune serum subsequently may prevent the growth and multiplication of virus in those cells.

The material used in the following cases was from two sources. A comparatively small portion was prepared by and purchased from the Cutter laboratories. The other larger portions were prepared by Squibb from plasma obtained from the blood banks sponsored by the American Red Cross. The source of globulin probably has a considerable bearing on the results obtained because, as Stokes[6] has pointed out, the large size of the plasma pools developed by the Red Cross tends to assure a surprisingly uniform titer of antibodies against infectious agents endemic to the geographical area in which the blood donors reside. There were no planned controls in this series as the gamma globulin was given to patients seen in a routine office practice and the results obtained were not anticipated. In all, the cases studied totaled over 280 seen in a period of approximately a year and half.

MEASLES

In measles the data have been reviewed previously by a number of workers and their reports indicate that gamma globulin represents the most satisfactory biological as yet developed for passive protection of exposed susceptibles or for attenuation of this disease. It would be redundant on my part to report anything other than that gamma globulin was given to 70 exposed susceptibles and the results were approximately the same as reported elsewhere. No effort was made to treat patients in this series.

MONONUCLEOSIS

In line with reports given by Drs. J. H. Park[4] and David Greer[3] in 1947 on the successful use of plasma transfusions as a means of therapy in mononucleosis and virus pneumonia, gamma globulin was given to 11 patients who presented the clinical and laboratory findings of infectious mononucleosis. Of these, 8 received 2.5 cc. and 3 needed twice that amount on successive days before clinical response was noted. This clinical response consisted of a rather prompt return of temperature to the normal and cessation of symptoms. No follow-up laboratory work was done after the initial diagnostic studies.

PRIMARY ATYPICAL VIRUS PNEUMONIA

In the past year, 35 patients who presented history, symptoms, and findings, both physical and laboratory, of primary atypical pneumonia were seen. Their ages ranged from 6 weeks to 18 years. The duration of symptoms varied from one week to three weeks before serum therapy was instituted. The majority were given 2.5 cc. of gamma globulin on two successive days; occasionally a third dose of 2.5 cc. was necessary. In all cases the temperature curve began to decline in from twenty-four to thirty-six hours following the first injection and subsequent improvement was rapid. In evaluation of the results it should be pointed out that none of these cases could be considered even moderately severe, but gamma globulin did seem to shorten the expected course.

MUMPS

Globulin was used in 5 cases of mumps without any demonstrable effect. This result was to be expected as evidence is available that in amounts up to 50 cc. gamma globulin from plasma pools derived from the general population is not usually effective in protecting exposed susceptibles against mumps.

HOMOLOGOUS SERUM JAUNDICE

A 2½ months old infant with a furunculosis was given two ampules of globulin plus surgical drainage and penicillin. Rapid recovery followed, but two weeks later he was again hospitalized for jaundice, died, and

was given an autopsy. The clinical and anatomic diagnosis was "homologous serum jaundice" probably contracted because of plasma transfusions given when he was 1 week of age.

VARICELLA

Immune serum was given as a prophylactic measure against varicella to 11 children, of whom 6 were sibling contacts, and 5 were living and attending nursery school in an area in which chickenpox was rampant. The sibling contacts did not contract the disease. As far as it could be ascertained, the other 5 did, but were apparently protected for a period of about six weeks before developing the typical rash.

UPPER RESPIRATORY INFECTION

The greatest number of cases in which gamma globulin was used in the present series consisted of various types of infections of the upper respiratory tract. An effort was made to ascertain from history and physical findings and by laboratory means the presence of a preexisting allergy. No effort was made to evaluate the worth of gamma globulin as a prophylactic agent in these conditions although Adams and Smith[1] in 1947 concluded that human serum gamma globulin given in arbitrarily selected doses reduces the incidence of attacks of acute respiratory disease in adults by about 40 per cent with a parallel reduction in the severity of the individual cases. In this series it was assumed that symptoms of a rhinitis existing for a period longer than two weeks was due either to a nasal allergy or sinusitis or both, and an effort was made to classify the disease before treatment was instituted.

One ampule of gamma globulin was given to about 75 patients with symptoms and signs of a disease of the upper respiratory tract in which the presence of allergy as a complicating factor was not demonstrable. The ages of the patients varied from 2 weeks to 19 years. The duration of symptoms, which were either repeatedly recurrent or almost continuous, varied from two weeks to two months. Fifty patients were relieved of their symptoms in thirty-six hours or less, 19 were considered improved, and in the remaining 6 there was either no reduction of symptoms or contact with them was lost.

Ten patients who had been treated by myself and others for recurrent acute suppurative otitis media were seen toward the end of the winter of 1947-1948. Repeated courses of penicillin and sulfonamides were given to each, with transitory improvement in every case. Globulin was given as a last resort, even during an acute phase. In all 10 patients so treated 2.5 cc. of globulin produced a cessation of suppuration and no recurrence to date.

Fifty patients in whom allergic rhinitis was believed to be the underlying condition were given globulin during the period of an acute exacerbation. The results in this group appeared to be directly proportional to the degree of their allergic control. In those children in whom the offending allergen was known and eliminated, the infectious phase of their respiratory difficulty was improved by a single injection of globulin. In those in whom all the allergens were not known or in whom control was poor 50 per cent showed considerable improvement for a period of about one month and the remainder received no discernible improvement.

PREMATURE INFANTS

Gamma globulin was given to 15 premature infants with the hope that early fortification with antibodies would lessen the likelihood of infection. The series was too small, the results too varied to draw any conclusions; however, Sweet studied the prophylactic use of globulin in a large series of premature infants and concluded that it did not materially affect incidence of infection or the gradient of the weight curve.

PERTUSSIS

Hyperimmune human serum globulin, which is prepared by inoculating prospective donors with pertussis vaccine, was given as a therapeutic measure to 20 patients with pertussis. The ages of these patients ranged from 3 weeks to 6 years; duration of symptoms before institution of therapy was from three to six weeks. The results were uniformly good, but were directly proportional to the ratio between the amount given and the weight of the patient and duration of the disease. The average total dose in these cases was 5 cc. or 2 ampules.

COMMENT

The case of homologous serum jaundice is included in this paper because gamma globulin has been suggested as a prophylactic agent in such conditions and also because, so far as I know, the 3 month old patient is the youngest on record proved to have this disease.

If the physician reviews the literature on such diseases as primary atypical or virus pneumonia and the various forms of disease of the upper respiratory tract, he becomes aware of the paucity of help offered other than palliative treatment in these common conditions. Gamma globulin, although certainly not a specific. seems to offer a more

direct method of attack at these comparatively mild but distressing conditions.

It is realized that from a purely scientific viewpoint, the results observed in this series are questionable because of the lack of control studies, but from the standpoint of a private practitioner immune serum prophylaxis and therapy seems to offer a weapon the true worth of which should be considered. It is hoped that more controlled studies will be done by those in a better position to do so than a pediatrician in private practice.

SUMMARY

1. A plea is made for more studies on the value of immune serum globulin in prophylaxis and treatment of certain infectious diseases.

2. A brief definition and the probable prophylactic and therapeutic action of gamma globulin are given.

3. A resume of the probable behavior of viruses in a susceptible host is presented.

4. Observations are made on the use of gamma globulin in more than 280 patients seen in a private pediatric practice within a period of a year and a half.

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ABSTRACT OF DISCUSSION

Dr. Sidney R. Kaliski, San Antonio: We are living in an era of dramatic and romantic therapeutic advances, among which the use of blood fractions for the prevention and treatment of disease is among the most important. Dr. Frawley's report is indeed advanced and provocative in that it includes the use of gamma globulin in diseases for which this substance has not as yet had general application and in which its efficacy must be proved or disproved by many well-controlled cases subjected to critical analysis.

Of particular interest are Dr. Frawley's observations in infections of the upper respiratory tract and otitis media, because the capricious course of these infections makes evaluation of treatment especially difficult. It is to be hoped that the essayist's plea for more studies on the use of immune globulin for those infections in which it has not heretofore had wide application will rapidly widen our experience and put immune globulin on a firm basis.

The pioneer observations of Drs. Park and Greer, employing pooled serum for infectious mononucleosis, and Dr. Frawley's observation that immune globulin acts similarly are valuable forward steps. Since globulin is administered intramuscularly, it may well be that it will prove superior to the intravenously administered serum because the possibility of homologous serum jaundice will thereby be avoided.

LYMPHOCYTIC CHORIOMENINGITIS

CLINICAL, EPIDEMIOLOGIC, AND LABORATORY ASPECTS

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The "infectious encephalitides," a designation recommended for all neurotropic virus diseases until an etiologic diagnosis has been made, embrace a number of nosologic entities which often constitute a perplexing problem for the medical practitioner. The present report is concerned with the clinical, epidemiologic, and laboratory aspects of one of these diseases.

A number of descriptive terms including acute serous meningitis, idiopathic meningitis, acute benign lymphocytic meningitis, and others have been used to designate a form of meningitis usually characterized by a lymphocytosis in a spinal fluid free from bacteria. In 1925, Wallgren[17] first described the clinical features of this disease and suggested the name "acute aseptic meningitis." Little advance was made until 1934 when Armstrong and Lillie[2] isolated an agent from the brain of a patient who presumably had suffered a fatal attack of St. Louis encephalitis. The same agent, which they labeled "the virus of experimental lymphocytic choriomeningitis," was later recovered from the spinal fluid of a laboratory worker, and has since been isolated by a number of investigators.[10] It is clearly evident, therefore, that a viral agent is the cause of at least some cases of so-called "acute aseptic meningitis."

CLINICAL ASPECTS

Lymphocytic choriomeningitis, now recognized as a distinct clinical entity, is an acute infectious disease which presents a remarkably variable clinical picture. The meningeal form, which may or may not be preceded by a "grippe-like" disease, is characterized by the appearance of fever and meningeal symptoms such as headache, stiff neck, vomiting, and positive Kernig and Brudzinski's signs. The cerebrospinal fluid, which is usually clear and free from bacteria, shows a pleocytosis ranging from a few cells to from 2,500 to 3,000 cells, predominantly lymphocytes. The protein in the spinal fluid is usually slightly increased, but the sugar and chloride content does not vary from normal. The disease, which is self-limited, lasts from several days to about one month, and recovery takes place without any residual effects. The virus of

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lymphocytic choriomeningitis may affect not only the central nervous system but other tissues as well. Widespread serologic surveys have indicated that approximately 11 per cent of blood sera from about 2,000 individuals selected at random from various parts of the United States contained antibodies specific for the virus of lymphocytic choriomeningitis.[18, 19] These studies indicated the existence of a type of infection which leads to specific immunity but without producing central nervous system symptoms, since many of those whose sera contained antibodies denied a history of any central nervous system disease. Subsequently, Armstrong and Hornibrook[1] reported the first proved case of systemic type of lymphocytic choriomeningitis virus infection. The patient was a laboratory worker engaged in lymphocytic choriomeningitis research at the National Institute of Health. Lepine and his associates[8] had previously demonstrated that human beings inoculated with a strain of lymphocytic choriomeningitis virus isolated from mice do not always develop meningeal signs although they do develop antiviral antibodies in their serum on recovery.

EPIDEMIOLOGY

The virus of lymphocytic choriomeningitis has been demonstrated under natural conditions in man, monkeys, gray mice, albino mice, and dogs. The widespread occurrence of the virus, together with the absence of evidence of the communicability of the disease from person to person, suggested the existence of some animal reservoir from which man may be infected. Armstrong and his associates[3] have presented well-documented evidence to show that the gray mouse may serve as a reservoir for the infection. The virus was isolated from pooled emulsion of liver and spleen of mice trapped in households where patients with the disease had lived. These observations have been confirmed by other investigators.[7] More recently, Dalldorf and his colleagues[6] reported cases of choriomeningitis among residents of a multiple family dwelling which harbored infected mice. According to these investigators, the incidence of lymphocytic choriomeningitis virus carriers among house mice in greater New York was found to be approximately 4 per cent.

The disease in man has been characterized by sporadic occurrence, that is, single cases without evidence to support infection by contact. Infection by contact or feeding, however, was suggested by the demonstration that virus is present in the urine and nasal secretions. The natural route for the transfer of the agent from animals to man is unknown. The epidemiologic data referred to above which suggest an association between many of the proved cases and house mice point to the mouse excreta as the source responsible for the transfer of the virus to man.

No vector has been found which harbors or transmits the virus of lymphocytic choriomeningitis under natural conditions. Experimentally, however, it has been shown that mosquitoes, ticks, and bed bugs can transmit the infection.[5, 9, 11] Since the majority of cases occur during the winter and spring months of the year, both the mosquito and the tick can be excluded as possible natural reservoirs because of their limited seasonal occurrence. Syverton and his colleagues[16] recently showed that the parasitic nematode Trichinella spiralis may serve as an effective vehicle for the experimental transmission of lymphocytic choriomeningitis virus from one host to the next. They do not claim, however, that their experimental data have any practical implication in the epidemiology of human lymphocytic choriomeningitis but speculate that the virus probably has a rodent reservoir of cosmopolitan distribution comparable to that of Trichinella. The role that a parasitic nematode can play during inter-epidemic periods as a reservoir for the maintenance of a virus under both experimental and natural conditions has been thoroughly investigated by Shope,[12] and has been reviewed in this JOURNAL by one of us.[15]

DIAGNOSIS

In differential diagnosis the physician must take into consideration a number of diseases which may present similar clinical manifestations: (1) acute aseptic meningitis, a disease similar to lymphocytic choriomeningitis but of unknown etiology; (2) tuberculous meningitis; (3) influenza; (4) syphilitic meningitis; (5) torula histolytic meningitis; (6) poliomyelitis; (7) postinfectious encephalitis; (8) other virus encephalitides.

Since lymphocytic choriomeningitis in its various clinical forms may simulate these other ailments, an etiologic diagnosis must depend upon specialized laboratory procedures. This may involve either recovery of the virus by inoculation of suitable laboratory animals with blood or spinal fluid drawn early in the disease, or by immunologic tests carried out on blood serum obtained during the early stages of the disease and again after recovery of the patient. The presence of specific neutralizing or complement-fixing antibodies is of particular significance if blood specimens taken during the acute phase are negative and become positive dur-

ing convalescence of the patient. Because of the specialized nature of these procedures, they have not been recommended for use in the general laboratory.

The virus of lymphocytic choriomeningitis is most readily recovered from the spinal fluid or blood drawn during the height of the attack. The agent has been occasionally isolated from the patient's urine and nasal discharges. Inoculation of susceptible animals must be carried out promptly, but if delay is unavoidable, specimens may be kept in a deep-freeze storage cabinet. Virus neutralizing antibodies are usually not demonstrable in the blood before eight to ten weeks following the onset of symptoms, and under rare circumstances may not appear. However, once neutralizing antibodies appear in the serum they tend to persist for months or even years. Complement-fixing antibodies, on the other hand, make their appearance earlier and tend to be less persistent than neutralizing antibodies.

CASE REPORT

R. P., a 4 year old white boy, was admitted to Parkland Hospital on June 20, 1947. He had been well until twenty-four hours prior to admission. At that time he started whining and complaining of pain in the roof of his mouth. During the night he developed fever and vomited three times. He became drowsy and seemed to doze off but could easily be awakened. Upon examination the patient was a well-nourished child apparently not seriously ill. The neck was rigid and the Kernig sign was positive. The deep and superficial reflexes were present and equal on the two sides. The patellar reflexes were hyperactive. There was a moderate hamstring spasm. The temperature was 102.4 F. on admission. The leukocyte count was 9,200 with 8 bands, 48 segmented cells, and 44 lymphocytes. There were 12.3 Gm. of hemoglobin. Urine was normal. A lumbar puncture showed an initial pressure of 150 mm. The spinal fluid, though clear, contained 783 cells per cubic millimeter, 99 per cent of which were lymphocytes. No organisms were seen on microscopic examination and aerobic and anaerobic cultures were subsequently negative. The Pandy reaction was slightly positive. The total protein was 69 mg. per 100 cc., sugar was 56 mg. per 100 cc., and chlorides, 119.4 mg. per 100 cc. The Wassermann reaction was negative.

The patient improved quickly following the initial spinal puncture. The temperature returned to normal within three days. Twenty-four hours after admission the spinal fluid contained 765 cells, approximately 95 per cent of which were lymphocytes. A spinal fluid specimen obtained on the day of admission, which showed no organisms on microscopic examination and by aerobic and anaerobic culture, was stored in the deep freeze storage cabinet at —76 C. for subsequent virus study. Twelve days after admission the child was discharged from the hospital in good condition and entirely free from symptoms. The spinal fluid at the time of discharge was negative. Three blood specimens for serologic analysis were obtained on the day of admission, nine days later, and three and one-half months after the onset of the disease.

On June 21, 1947, the spinal fluid referred to above was inoculated intraperitoneally, intracerebrally, and subcutaneously into each of 4 Swiss albino mice. Six days later 1 animal became ill with evidence of central nervous system symptoms and was killed for serial passage of brain tissue. Two mice became ill eight days after inoculation with evidence of central nervous system symptoms. These animals were killed and the brains were stored in the deep freeze storage cabinet. The fourth mouse developed no symptoms and was discarded after an observation period of one month. None of the animals which received intraperitoneal, intracerebral, and subcutaneous injection of heparinized blood obtained on the day of admission developed signs or symptoms of encephalitis. The agent which caused symptoms in the first mouse six days after inoculation was passed through six serial passages in white Swiss mice by intracerebral inoculation. With each passage, aerobic and anaerobic cultures proved negative. Identification of the agent as the virus of lymphocytic choriomeningitis was made by neutralization tests with the serum of monkeys immunized with known strains of this virus. Most of the animals showed symptoms of tremor and staring coat from five to six days after inoculation, while some animals developed convulsions and died from seven to nine days following inoculation. In some instances characteristic extension and rigidity of the hind legs was noticed at the time of death. To identify the agent further, cross protection tests were conducted using the W. E. and J. P. strains of lymphocytic choriomeningitis virus, kindly provided by Dr. Gregory Shwartzman and Dr. Albert Milzer, respectively.

The presumptive clinical diagnosis of acute lymphocytic choriomeningitis was confirmed in the virus laboratory by demonstrating the presence of neutralizing and complement-fixing antibodies in blood serum obtained during convalescence. The intervals from the onset of symptoms to bleeding for convalescent serum were nine days and three and one-half months. The antigen for the complement-fixation test was prepared from infected mouse spleen according to the method of Smadel and his associates.[14] The neutralization tests were performed according to the method described by Simmons and Gentzkow.[15] The W. E. strain of lymphocytic choriomeningitis virus was used in these experiments. The intracerebral route of inoculation was used. The convalescent phase serum specimen obtained nine days after onset of the disease showed a neutralizing index* of 16, while the specimen obtained three and one-half months after onset showed an exceedingly high neutralizing index, less than 200. The serum obtained nine days after onset, in a dilution of 1:32 fixed complement in the presence of antigen prepared with the W. E. strain. The serum obtained three and one-half months after onset when mixed with antigen prepared from the same strain fixed complement in a serum dilution of 1:8.

SUMMARY

1. The clinical, epidemiologic, and laboratory aspects of lymphocytic choriomeningitis are presented.

2. There is reported the case of a 4 year old boy in which the clinical diagnosis of acute lymphocytic choriomeningitis was confirmed by recovery of the virus and by immunologic tests carried out on blood serum obtained during the early stages of the disease and again after recovery of the patient.

*The neutralizing index refers to the amount of antibody in the serum being tested which is sufficient to reduce the titer of the virus, as compared with the virus titer in the presence of normal control serum or serum obtained during the acute phase of the disease.

3. Since lymphocytic choriomeningitis in its various clinical aspects may simulate other ailments, an etiologic diagnosis must depend upon specialized laboratory procedures.

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ABSTRACT OF DISCUSSION

Dr. Elias Strauss, Dallas: It has been my experience that the clinical diagnosis of lymphocytic choriomeningitis is made too frequently. In 1925 Wallgren proposed the term "acute aseptic meningitis" to cover cases of acute meningitis of nonbacterial origin, characterized by a mononuclear cellular reaction in the spinal fluid and a good prognosis. The virus of lymphocytic choriomeningitis produces an illness of this type but it is by no means the only, or the most common, agent which does so. The clinical diagnosis of lymphocytic choriomeningitis is too often made when, in fact, the only justifiable clinical diagnosis that can be made is "acute aseptic meningitis of unknown etiology." Only by appropriate laboratory tests can an etiologic diagnosis be established. Unfortunately, such diagnostic procedures are still beyond the scope of routine diagnostic laboratories.

In addition to lymphocytic choriomeningitis, a considerable number of other viruses produce an illness falling into the general clinical classification of acute aseptic meningitis. Among these may be mentioned mumps, infectious mononucleosis, lymphopathia venereum, herpes simplex, poliomyelitis, equine and St. Louis encephalitis. Epidemiologic considerations and associated clinical findings are helpful in pointing the way toward a correct etiologic diagnosis in some instances. In addition to all of these agents, however, there are still unknown agents, presumably viruses, which cause the clinical picture of acute aseptic meningitis. In the experience of a number of laboratories (Dr. Sulkin and I at Southwestern Medical College have had a similar experience) an etiologic diagnosis cannot be made in the majority of the sporadic cases of acute aseptic meningitis even after elaborate laboratory study. In the past year 12 sporadic cases of acute aseptic meningitis were studied; in almost every case the clinical diagnosis of lymphocytic choriomeningitis had been made, yet in only 3 of the 12 cases was this diagnosis confirmed. In the other 9 cases no etiologic diagnosis could be established.

Are there any clinical features which might enable physicians to suspect lymphocytic choriomeningitis and separate it from the larger group of aseptic meningitides? In the descriptions of proved cases of lymphocytic choriomeningitis, three features stand out: (1) There is sometimes a recognized association with ordinary house mice, although actual handling or contact with mice is rare. (2) There is often a prodromal febrile illness of nonspecific "grippe-like" nature lasting from one to two weeks followed by a period of one or two days of well-being which precedes the onset of meningeal symptoms. (3) The cell count in the spinal fluid is often 600 or more per cubic millimeter. I do not wish to state that these are invariable features of lymphocytic choriomeningitis, but they are more likely to occur in proved infections with this virus than in the larger group of unknown etiology. If the physician restricts the clinical diagnosis of lymphocytic choriomeningitis to cases with one or more of these features, he will make the clinical diagnosis less frequently but will be more likely to have his diagnosis confirmed by the laboratory.

Dr. J. P. Gibson, Abilene: Which is preferable, blood or spinal fluid examination?

Dr Orion Thompson, Tyler: What is the treatment?

Dr. Brooksaler, closing: Both spinal fluid and blood should be submitted for laboratory study. There is no specific treatment.

ARTIFICIAL KIDNEY

An artificial kidney developed by three researchers of Toronto, Canada, Gordon Murray, M.D., Edmund DeLorme, M.D., and Newell Thomas, is described in the August 28 issue of *The Journal of the American Medical Association.*

The apparatus, which will reproduce the function of the kidney and will remove many, if not all, of the toxic substances in the blood, has been successfully used on patients and several days of treatment cause no ill effects, the researchers report.

The artificial kidney operates by means of a cellulose membrane which filters the blood, separating large particles from small ones. This membrane is placed in a chemical bath containing sodium chloride, other chlorides, sodium phosphate, sodium carbonate, and dextrose. A pump removes blood from the large trunk vein of the body which provides circulation to the legs and passes it into a tube, through the membrane, and into the opposite vein of the flank. As the blood of a patient circulates through this route, the toxic substances fall out through small openings of the membrane. Large molecules such as proteins and hormones are carried back into the blood stream.

PATHOLOGIC CONDITIONS OF THE NASOPHARYNX

DIAGNOSIS AND TREATMENT

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No area in the upper respiratory system has had more attention than the nasopharynx in the last decade. Before this period it was one of the most neglected, probably because of its inaccessibility to examination. This region is subjected to all the changes in temperature and changes in humidity as well as purulent secretions that flow over it when nasal disease or dysfunction is present.

In 1938, Carmody[2] read a stimulating paper entitled "The Epipharynx—The Almost Unknown in Oto-Laryngology" which must have acted as the spark plug, for increasing interest in this field of research and literature has manifested itself. Crowe[3] and his associates have further stimulated interest by their introduction of the comparatively simple method of dealing with hypertrophied lymphoid tissue about the eustachian orifices.

DIAGNOSIS

The problems encountered in diagnosis of disease in the nasopharynx are not really great, but skill, patience, and persistence are required sometimes. I dare say most of the errors in diagnosis of disease in the nasopharynx lie, not in the ignorance of disease in this location, but rather in carelessness of examination. In a routine examination of the ear, nose, and throat the problems encountered sometimes in visualization of the nasopharynx encourage its examination to be foregone until another day and forgotten.

It might be said that any symptoms above the shoulders may be a direct or indirect result of disease within the confines of the nasopharyngeal region. Symptoms relative to such disease therefore are many[5]: the burning sore throat occurring with the onset of an acute rhino-pharyngeal infection; the localized nasopharyngeal infection with pyogenic organisms producing ulceration; the diphtheritic sore throat uncommonly limited to this region; the tertiary syphilitic sore throat also uncommon; stopped ears due to benign and malignant tumors, enlarged adenoids, or edema about the eustachian orifice; nasal obstruction due to congenital or acquired atresia of the chonae, atresia of the nasopharynx, nasopharyngeal polypi, tumors, both benign and malignant, hypertrophied adenoids, and cysts. Hemorrhage from the nasopharynx, while rare, may indicate a malignancy. Extension of malignant tumors into the base of the cranium may be manifested by involvement of the second, third, and fourth nerves, both the second and third branches of the fifth nerve, the sixth cranial nerve, the gasserian ganglion, and the sella tursica producing pain in the eye, trifacial neuralgia, and other symptoms depending on the nerve or organ involved. Invasion may extend to the foramen lacerum and the jugular foramen involving the ninth, tenth, eleventh, and twelfth cranial nerves to produce paralysis of the corresponding half of the palate, tongue, pharynx, larynx, the sterno-cleido-mastoid, and the trapezius muscle. The juvenile type of fibroma usually causes nasal obstruction. The pressure exerted by their growth may erode bone and extend into the orbit and cause proptosis, and even sometimes into the cranium, producing some of the above symptoms and terminating fatally in meningitis.

Local manifestations in the nasopharynx occur in systemic disorders. There is no definite diagnostic criteria but the physician cannot help but be impressed when he finds disease without a local explanation. While vitamin deficiency may be tremendously exaggerated, I am convinced of its importance in some recurrent and frequent attacks of nasopharyngitis. Allergy of the nasopharynx, while rare, does occur, but it is more frequently associated with nasal allergy. I know of no better way to make the diagnosis than by the usual method of elimination. As in all other allergies it is important to convince the patient of the procedure necessary and secure his utmost cooperation or the diagnostician will surely fail. Faulty digestion due to the lack of some of the juices in the stomach, principally hydrochloric acid, seems to cause considerable disturbance in both the nose and nasopharynx. I have seen some startling results when this condition was corrected.

Nasopharyngitis secondary to nasal disease, such as the crusting associated with ozena or atrophic rhinitis, and pharyngitis sicca are more likely to be part of the disease than a complication.

Because of the variety of these symptoms, findings, and diseases the nasopharynx must be examined carefully on every patient who presents himself with symptoms in relation to the ear, nose, and throat. A good study of the nasopharynx can be made in a few seconds on the majority of patients by using a small mirror placed in the back of the throat. I find myself frequently trying to examine the nasopharynx with a dull mirror. After being boiled several times, mirrors always become dull, and the contrast and clearness

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of vision is remarkable when a new one is used. When a nasopharynx can be examined well with a small mirror, there is nothing that can take its place, not even the nasopharyngoscope; yet there is nothing that can take the place of the nasopharyngoscope when a patient has a shallow nasopharynx, a palate close back against the posterior wall, or an unruly gag reflex. While a good mirror study will give a rather complete picture, the study is never complete until the nose is well shrunk and the nasopharynx seen through anterior rhinoscopy. With a little pontocain spray, a little patience, and perseverance, I believe a good study of the nasopharynx can be made in all adults and most children more than 5 or 6 years of age with mirrors or the nasopharyngoscope. A biopsy specimen must be taken at times to complete a diagnosis, and most are not difficult to secure. They can be taken through the nose sometimes when they occur in the region under direct vision by anterior rhinoscopy, and from the lateral walls and the lower part of the nasopharynx by elevating the palate with the Love retractor or the Yankhauer speculum.

My greatest problem in making diagnosis of lesions in the nasopharynx is in children less than 4 or 5 years of age. Most of them are not cooperative and I almost never feel that I have a good visual picture until the patient has been examined under a general anesthetic. Of the less complicated procedures, I believe that I learn more from anterior rhinoscopy when the nose is well shrunk than from any other single procedure. It is imperative to have a strong light with parallel rays and a nose exceptionally well open. Fortunately, about the only diseases found in children are acute infections, hypertrophy of the adenoids, congenital defects of the posterior nares, and juvenile fibroma. Holinger has devised a new scope with a distal lighting to be used through the mouth which has some advantages. The Yankhauer speculum and other similar instruments offer about the only method of examination short of general anesthetic. Digital examination in infants up to 3 or 4 years of age gives some information, but it does not take the place of visual study.

TREATMENT

The problem of treating diseases of the nasopharynx is not always simple. Many cases call for purely medical treatment. All laryngologists have pet treatments for the early acute condition of the nasopharynx recognized more by the symptom known as the "hot spot" in the throat than any other way. Usually this is due to drying of the pharyngeal mucosa and the physiologic function may be reestablished before the infection is well developed by some mild counter irritant and saline nose drops. The ulcerative nasopharynx, practically always due to pyogenic organisms, responds well to the sulfa drugs and penicillin; the diphtheritic lesion to antitoxin; and the syphilitic lesions to neosalvarsan and the more modern treatment of penicillin.

Nasopharyngeal lesions secondary to nasal disease usually cannot be improved until the nasal physiology has been reestablished. Atrophic conditions of the nasopharynx usually occurring along with ozena and atrophic rhinitis are best relieved by giving large doses of estrogenic hormone. Only temporary improvement, and hardly that, can be expected by the local application of estrogen in olive oil.

The treatment of an allergic nasopharynx (incidentally, nearly all have secondary infection) lies primarily in the diagnosis. Of course the best treatment is the removal of the allergen from the environment of the patient. I am convinced that there are certain foods which in some persons cause an allergic nasopharyngitis not accompanied by the usual allergic rhinitis. I know of no better way than to begin with the more common foods which are known to be responsible for most allergies and eliminate them one at a time, the proof finally being the recurrence of the symptoms when the food is eaten in substantial quantities. I cannot pass without mentioning those cases of nasopharyngitis due to drugs such as the common nose drops, penicillin, and streptomycin. I have 2 patients in whom the nasopharynx becomes much worse after the use of ictholdine, presumably because of a sensitivity to the iodine.

Nasopharyngeal polypi offer no particular problem although I have seen it necessary to do a submucous resection to get at the attachment in the middle meatus. Nearly all of these polypi arise from the maxillary antrum, are single, often firm and fill the nasopharynx. Rarely, if ever, have I tried to remove one by using a snare in the nasopharynx. I find it much easier to cut the attachment in the nose and remove the polypus either through the nose or if it is too large, with curved forceps by way of the mouth.

Juvenile fibromas of the nasopharynx are notorious for their capacity to bleed severely, sometimes almost uncontrollably, when attempts have been made to remove them surgically. The use of radium needles, radon seed, and electrocoagulation seems to be effective. If a reasonably positive diagnosis can be made without biopsy, it should be

done. Severe hemorrhages have occurred from even this simple procedure. Figi[4] reported the use of electrocoagulation and radium either in the form of needles or the T radium applicator. Since 1924 electrocoagulation has been used in about half the cases but not without difficulties from severe hemorrhage.

Cysts of the nasopharynx are of two types, the retention cyst of the adenoids and vestigials of the pharyngeal bursa, both of which can be easily removed by surgery.

Malignancies of the nasopharynx are usually proved by biopsy. New,[7] however, reported he was unable to get a positive biopsy the first time in 37 out of 271 cases of malignancies of the nasopharynx. Of these, grade 4 squamous cell carcinoma was the most common, lymphosarcoma the next most common tumor. Adenocarcinoma, fibrosarcoma chondrosarcoma, myxosarcoma, and hemangio-endothelioma are rarely seen. Schmincke tumor[1] or lympho-epithelioma of the nasopharynx, while rare, has been reported repeatedly. Roentgen therapy and radium introduced into the nasopharynx by means of a T applicator is the most satisfactory treatment, but five year cures are rare. Electrocoagulation has no place in treating these malignancies.

Atresias of the nasopharynx divide themselves into two types, the acquired, which sometimes follows syphilis or poorly done tonsillectomies, and the congenital.

The acquired form, particularly if there is normal mucous membrane below the stricture, can be more effectively treated after the method of MacKenty.[6] In the congenital form a membrane usually extends from the posterior aspect of the soft palate to high up on the posterior nasopharyngeal wall. By punching out the membrane and treating the edges with monochloracetic acid, a good opening may be maintained.

Congenital atresia of the posterior nares are nearly always bony and are frequently bilateral. When they are seen in the adult patients the problem is not so great, but when seen in the newborn, the difficulties are almost insurmountable, because of the small size of the structures. In 4 cases that have come under my observation, 2 were congenital and 2 were acquired. One of the congenital cases was bilateral, the other unilateral, both in adults. They were given adequate airways by drilling an opening through the bony septum and punching out the remaining bone with a sphenoid punch. In the case with bilateral obstruction, a submucous resection was done and the septal process of the vomer removed. The 2 cases of membranous septa were treated by electrocoagulation. Ruddy[9] described a transpalatine approach for restoring the airways in infants which has proved successful.

The treatment of lymphoid tissue of the nasopharynx deserves considerably more discussion than can be given it here. While the problem may appear simple, it is not entirely so because of the not too infrequent recurrence of large masses of lymphoid tissue in the nasopharynx after adenoidectomy. There are two reasons for this, (1) a definite recurrence and (2) tissue high up in the vault was not removed. Before the adenoids are removed at surgery, the palate should be retracted, the area inspected, and the region palpated. This procedure should also be repeated after the removal has presumably been accomplished and before the patient is allowed to leave the operating room. I do not see the necessity, however, of removing every vestige of lymphoid tissue, for too radical removal will result in scar tissue replacement, a poor substitute for normal tissue. If the lymphoid tissue is too widespread, that which is in folds and is more or less pedunculated should be removed, leaving the flat tissue to the radium applicator.

So much discussion has taken place in the last few years at state and national meetings regarding the radium application that I doubt much need be said about it. I think most otolaryngologists have come to use it because of the localized effect. We do not have the fear of damaging the growing structures and ossification centers in children, as might be the case with the use of roentgen-ray over the same region.

There is no doubt that the radium application has a limited therapeutic value. Proctor[8] has shown that the monel metal applicator is best, because it emanates the therapeutic beta rays more effectively without the secondary radiation. Most of the rays are absolved in the first 3 mm. of tissue.

There is still a place for the use of roentgen-ray in treating lymphoid hyperplasia when it is suspected higher up in the tube, for enlarged peritubal glands, or widespread recurrence of lymphoid tissue in the nasopharynx.

SUMMARY

1. Adequate routine examination of the nasopharynx is encouraged.

2. Biopsies even in definite malignancies are not always positive and should be repeated until conclusive.

3. Many of the recurrent cases of nasopharyngitis are explained on the basis of allergy, deficiency in gastric juices, and vitamin deficiency.

4. Use of the radium applicator is encouraged, but at the same time it should not be abused.

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ABSTRACT OF DISCUSSION

Dr. Wallace B. Wilkinson, Dallas: I agree with Dr. Harris that the region of the pharynx generally called the nasopharynx or the epipharynx nas been often discussed in recent times, but it is still too frequently the forgotten area of the pharynx. Most of this neglect is due simply to failure to make a complete examination of the nose and throat. Too often unless the patient's symptoms and complaints point directly to this area of the pharynx the examiner does not go to the trouble properly to see this area. An examination of the pharynx is not complete without a good view being obtained of all parts of the nasopharynx. This is as important in relation to the pharynx as a good view of the anterior commissure is important in the examination of the larynx.

The nasopharynx is involved in practically every case of pharyngitis regardless of the etiologic condition. It is too often overlooked that the nasopharynx is involved to some extent in every nasal condition that presents itself. There is no part of the nose and throat that gives more definite signs and indications of what is taking place in the nose above, than does the area of the nasopharynx.

The dryness of the mucosa in pharyngitis sicca and similar atrophic conditions of the nose are sometimes first seen in this area.

The moist edematous appearing mucosa with mucous postnasal drainage without inflammatory reaction in the nasopharynx is always suggestive of an allergy. Dr. Harris brought out the possibility of food allergies. I would like to add the one other type of allergy that in my experience has been most often the offender when symptoms are present in the nasopharynx. Many patients have had definite reactions to house dust and so many have improved under treatment for that type of allergy that in my mind house dust has become the number one offender in allergies of the nose with troublesome "postnasal drip" and nasopharyngeal irritation. It is my strong contention that much more can be done for these patients by close cooperation between the otolaryngologist and the allergist than can be done by the otolaryngologist alone.

The red, thickened, lateral pharyngeal bands with which otolaryngologists are too familiar is frequently an indication of an infective type of postnasal drip, probably from some of the posterior sinuses.

One other condition which always results in a diseased state of the nasopharynx and which Dr. Harris failed to mention is acute mononucleosis. This acute systemic disease, characterized by extremely high temperature, a distinctive ulcerative nasopharyngeal lesion, and a characteristic odor is often missed at diagnosis because its real pathology is located above the palate. There it is missed in a routine examination of the pharynx until, after no improvement has been obtained with chemotherapy, an adequate nasopharyngeal examination is finally done.

I would like to reemphasize the need of a good examination of the nasopharynx following adenoidectomy. The removal of the adenoids has too often become a more or less routine part of a tonsillectomy. It is surprising what large pieces of lymphoid tissue can be missed by the operator unless such an immediate view of the nasopharynx is obtained. Also let me stress that it is not necessary to dig so deeply that the tissues are removed down to the periosteum. It is a mistake to attempt to remove the whole thickness of the mucosa in an effort to remove all lymphoid structures. If the surface is smooth and all chronic infection in the nose above is taken care of, there will be few cases of secondary hypertrophy of the adenoids. It would seem that much damage might be done by removing the whole thickness of the mucosa. I agree with Dr. Harris that in adenoidectomy the surgeon should study under direct view the area of the nasopharynx before the patient is allowed to leave the operating table.

I believe Dr. Harris has passed too lightly over the use of the radium applicator in the treatment of lymphoid tissue about the orifice of the eustachian tubes. It is true that this therapy is not a cure-all; however, it is the first time otolaryngologists have had anything at all good to offer those hard of hearing children whose obstructive deafness seems to be caused by blockage of the tubes. The monel metal radium applicator will definitely cause the destruction or recession of lymphoid tissue. Its proper use is harmless and in a good percentage of cases there is a noticeable improvement of hearing. The best results are obtained when treatment is instituted before hearing has been impaired for too long a period. Results cannot be expected except in cases where there is real hypertrophy of lymphoid tissue at the orifice of the eustachian tubes. Cases should be carefully selected.

THEPHORIN FOR HAY FEVER

Clinical tests of a new drug, thephorin, have shown it to be more than 90 per cent effective in treating hay fever and other allergic conditions, according to an article in the June, 1948, *Illinois Medical Journal.*

Reporting on 142 allergy cases treated with the drug, John Peters, M.D., of Oak Park, disclosed that thephorin brought good results in 129 cases, or almost 91 per cent. The drug was especially effective in hay fever, good results being obtained in 66 of 68 patients treated, or 97 per cent. Similarly, in treating 34 cases of combined hay fever and asthma, he found that "thephorin was effective in controlling the symptoms of 91 per cent of the patients suffering from both hay fever and asthma," with 3 failures in the group. In 16 cases of asthma, Dr. Peters found thephorin effective in 75 per cent. "All of the failures occurred in bronchial asthma. The drug was 100 per cent effective in pollen and grass asthma."

Thephorin is the newest of the so-called antihistamine drugs, and, according to Dr. Peters, appears to be the least toxic of the antihistaminics. In the 142 patients, only 15 complained of side reactions, "or approximately 11 per cent of all cases." These side reactions included gastric disturbances in 5 patients; insomnia in 3; nervous or jittery feeling in 2; sweating in 2; and fatigue, sleepiness, loss of appetite, blood in urine, frequency of urination, labored breathing, and numbness of extremities in 1 patient each.

THROMBOPHLEBITIS OF CAVERNOUS SINUS WITH RECOVERY

J. A. STOOL, M.D. and R. D. LOMAS, M.D.
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Fulminating bacterial thrombophlebitis of the cavernous sinus prior to 1936 was almost uniformally fatal.[5] Those few patients who did recover may have had an orbital cellulitis and the accuracy of the diagnosis could be questioned.[10] Eagleton[3] listed the criteria for establishing the diagnosis of thrombophlebitis of cavernous sinus as (1) a known site of infection, (2) an infection of the blood stream, (3) early signs of venous obstruction, (4) involvement of nerves in the sinus, (5) a neighborhood abscess of the soft parts, and (6) symptoms of a complicating disease.

Most of the cases that have been reported had as the responsible organism Staphylococcus albus or aureus.[5] Since there are now three known antistaphylococcal agents, reports of recoveries from this malady should be increased. A survey of some of the recent literature has found this to be so. Agents that have been reported to effect these recoveries were sulfonamides, penicillin, and antistaphylococcal serum. In some cases sulfonamides alone were reported to effect cures[2, 6, 9] while in others sulfonamides were not of much benefit. In 1 case[1] thrombophlebitis developed a month after cellulitis of the face was treated with sulfathiazole. Goetzman and Friedman[4] reported a recovery using staphylococcus antitoxin. There have been several reports of recoveries with penicillin in recent years.[1, 11] Occasionally it is necessary to use all three bacteriostatic agents as is demonstrated by the case of Sears and Wilson.[8]

A further advance in the treatment of this disease was made by Schall[7] with the introduction of an anticoagulant, heparin, to hinder the further extension of the thrombosis. Most of the cases reported have received either heparin or dicumarol as part of the treatment. The feeling about anticoagulants in the treatment of this disease is that although there is some danger of excessive bleeding, especially in the brain, cavernous sinus thrombophlebitis is a desperate disease in which such an agent is justified.

FIG. 1. Photographs of a patient with thrombophlebitis of cavernous sinus before and after treatment.

Herewith is presented another case in which recovery followed the use of penicillin, sulfadiazine, and anticoagulants. For simplification the important features of the case have been included in figure 2.

CASE REPORT

On December 16, 1946, I. R., a 9 year old Negro boy, was admitted to the pediatric service of Hermann Hospital because of proptosis of the left eye and fever. Three days before admission the left eye became "swollen." The next day the patient complained of headache and pain in the abdomen. There was no history of cough or vomiting. On the third day the patient was brought to the Emergency Room, at which time he was reported as having a diffusely prominent left eye with a thick yellowish discharge. The right eye was normal but nuchal rigidity was noted. A lumbar puncture was performed; the fluid was clear and 3 to 5 white cells per cubic millimeter were found. Sulfadiazine 0.5 Gm. every four hours and a 5 per cent sulfathiazole ophthalmic ointment were prescribed.

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The patient was sent home, there being no hospital bed available. The next day, the temperature was 105.8 F. and the adnexa of each eye were swollen. The patient returned to the hospital and was admitted. Past history was not pertinent.

Physical examination at the time of admission revealed the patient to be semi-stuporous; his temperature was 105.8 F. He was obviously critically ill. The left eyelid was edematous; the eyeball was proptosed and the overlying structures were moderately tense. The pupil and iris did not seem to be involved. There was a moderate amount of mucosanguinous conjunctival secretion. Extra-ocular movements were limited. The right lid was slightly edematous and extra-ocular movements limited to a less degree. No view of the fundi could be obtained. Pitting edema was noted over the left mastoid process. Nuchal rigidity was pronounced; many large, firm, nontender lymph glands were palpable in all areas of the neck. Blood pressure was systolic 110, diastolic 55. A blowing systolic murmur was heard over the precordium. The chest was essentially normal as was a neurologic examination. No other physical findings of importance were noted.

On admission the white blood count was 18,000 with a moderate shift to the left. A lumbar puncture was again performed; the fluid was under no increased pressure and acellular. Staphylococcus albus was cultured from the blood.

The diagnosis of thrombophlebitis of cavernous sinus was made on admission and treatment was started. Penicillin was given intramuscularly at intervals of two hours. Two dosages were given, 30,000 units and 100,000 units as shown on the chart. Sulfadiazine was continued and, with the exception of 1,000 cc. of 1 per cent sodium sulfadiazine given intravenously on December 21, the dose was 1 Gm. by mouth every four hours. On the day of admission 100 mg. of heparin was given intravenously and 200 mg. of dicumarol orally. The next day 100 mg. more of dicumarol was given.

On the second day the patient was more rational but physical signs of a pneumonic process had become evident. Roentgenograms of the chest revealed bilateral broncho-pneumonia; views of the sinuses and mastoids were considered normal. At this time a slight weakness of the left facial muscles was noted; this disappeared within twenty-four hours.

It was decided to discontinue the dicumarol on December 18, because of conjunctival hemorrhages from the left eye. Although these hemorrhages were not of sufficient amount to cause anxiety, it was thought that clinical improvement was such that discontinuance would not retard recovery. The edema of the eyelids had now subsided sufficiently to enable visualization of the fundi. These were considered normal, except for questionable venous engorgement. A rough estimate of the patient's vision revealed 20/25 in the right eye and 20/70 in the left eye. Hematuria developed on the sixth day and the sulfadiazine was discontinued. The urine returned to normal within twenty-four hours. By the seventh day all findings had become localized to the left eye. Extra-ocular movements and corneal sensation, which had initially been impaired, were now normal.

Serial roentgenograms revealed the pneumonic process to be progressively clearing. On the twenty-sixth day penicillin was discontinued. No residua was noted with the exception of a mild proptosis of the left eye and ptosis of the left upper lid. During the remaining three days of hospitalization the patient was given ambulatory privileges. He encountered no difficulty and had no complaints. He was discharged on the thirtieth day.

A total of 19,310,000 units of penicillin was given and 46 Gm. of sulfadiazine.

Several follow-up examinations have been made since the patient's discharge from the hospital. The patient has had no complaints, but the mild proptosis of the left eye and ptosis of the left upper eyelid have continued. Vision and fundi have been considered normal.

SUMMARY

A case of thrombophlebitis of the cavernous sinus and complications with recovery is presented. Penicillin, sulfadiazine, and anticoagulants were used in the treatment. No attempt was made to evaluate the role of the drugs.

We wish to acknowledge the cooperation and suggestions of Dr. C. C. Cody III and Dr. Edward Garrett.

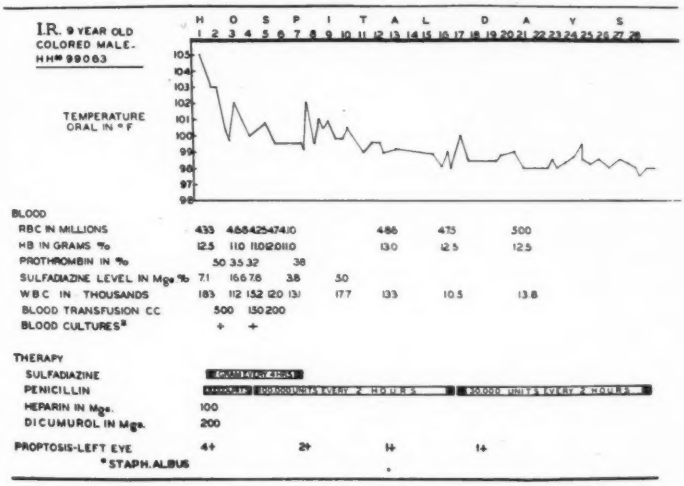

FIG. 2. Summary of a case of thrombophlebitis of cavernous sinus.

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ABSTRACT OF DISCUSSION

Dr. A. N. Champion, San Antonio: This paper shows the good results that may be obtained by the careful study and skillful treatment of a dangerous complication. With the aid of modern therapy and the early recognition of lesions that may lead to cavernous sinus thrombosis many cases can be prevented. It is well to remember that the main connections of this sinus are the ophthalmic veins, the pterygoid plexus, and the petrosal sinuses. Any infected area that these channels drain should be handled with the most gentle care. For example, it is dangerous to incise or squeeze a furuncle of the upper lip or nasal vestibule. Infections of the teeth, pharynx, or nasal sinuses should not be treated surgically until a delimiting fibrous wall has occurred. Fortunately, by the use of roentgen therapy, the sulfonamides, and antibiotics, many of these lesions will subside without drainage and most of the remainder can be drained safely.

If possible, the anticoagulants should be started early. Neither the sulfonamides nor antibiotics will have much influence on an impervious thrombus. It is safer to make frequent determinations of the prothrombin time so that spontaneous bleeding may be avoided. Large doses of vitamin K may be needed to combat that tendency.

Finally, diagnosis should be attempted before the full development of the criteria listed by Eagleton. With a seriously ill patient who has increasing orbital edema and chemosis and who has protrusion and limited movements of the eye, a provisional diagnosis of cavernous sinus thrombosis is justified. Careful roentgen ray and nasal examination should differentiate an orbital abscess. With the diagnosis made, the treatment outlined by the essayist should be followed.

SCHOLARS IN MEDICAL SCIENCE

Medical schools in the United States and Canada are invited by the John and Mary R. Markle Foundation to make nominations for the second group of Scholars in Medical Science on or before December 1, 1948. Each school, through the dean, may nominate one candidate. No nominations from individuals will be considered.

The program is designed to aid promising young men and women planning careers in academic medicine. They should have completed the usual fellowship training in some area of science related to medicine and should hold, or expect to hold, in the academic year 1949-1950 a full-time faculty appointment on the staff of a medical school.

Grants of $25,000, payable at the rate of $5,000 annually, will be made to the schools over a five-year period for the support of each scholar finally selected, his research, or both.

ARMY HOSPITAL BEDS FOR VETERANS

A total of 3,035 beds in Army hospitals throughout the United States have been allocated for treatment of veterans, Major General Raymond W. Bliss, Surgeon General of the Army, has announced. The allocations were made at the request of the Veterans Administration. Among the hospitals where veterans will be assigned beds are Brooke General, San Antonio, 400 beds, and William Beaumont General, El Paso, 150 beds.

MANAGEMENT OF PAROTID LESIONS

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Lesions of the parotid gland have long commanded a great deal of respect from the conscientious surgeon. The mental hazard to every operation in this region is, of course, the facial nerve, which transverses through the gland and eventually branches widely before its exit to supply the muscles of facial expression. For years, the tumor or parotid lesion was given primary consideration and the fate of the facial nerve was left largely to chance.

Recent strides in the technique of nerve grafting and nerve reconstruction have been advanced and perfected during the last war. With the increase in peripheral nerve study, the facial nerve has been of particular interest to physicians treating pathologic conditions of the face and ears. Especially has the facial nerve been studied with regard to its usual location, and better methods for its dissection and preservation have been advanced. The well trained surgeon today considers that a gross fault in technique has been committed if this facial nerve is cut or damaged during an operation in the region of the parotid gland or mastoid process. Often long and painstaking efforts are necessary to locate and preserve the facial nerve, but the results are well worth the task.

With these introductory remarks, let us consider various lesions of the parotid gland that confront the surgeon, and deal with some of the methods of therapy. A parotid lesion may be roughly classed in one of the following groups: (1) inflammatory lesions, (2) mixed tumors, (3) carcinomas, (4) cysts and adenomas, and (5) miscellaneous.

INFLAMMATORY LESIONS

Inflammatory lesions may be acute as in epidemic parotitis, general sepsis, and occasionally where a salivary calculus obstructs the duct. Such conditions readily lend themselves to a diagnosis through history of onset and the examination of the gland. The duct may be red and pouting, and frequently a purulent exudate can be expressed by milking the duct. The gland is quite tender on palpation. Hot compresses and roentgen therapy relieve pain and help with resolution of the gland. Cleanliness of the mouth is particularly important in such cases of general sepsis. The physician must never fail to combat the dehydration which may well be the instigating factor in certain cases. With adequate chemotherapy recovery can usually be hastened. Where calculi are present, their

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removal is essential for complete resolution of the gland. When suppuration occurs, adequate incision and drainage must be obtained to effect a cure. Chemotherapy is not a substitute when surgery is indicated.

Chronic inflammatory lesions of the parotid gland may offer more of a diagnostic problem. Some specific granulomas such as tuberculosis or actinomycosis should always be carefully considered as a possibility. A chronic inflammation of the parotid may offer real difficulties in distinguishing it from a neoplasm in the absence of general constitutional symptoms. The history of onset and the particular occupation of the patient should be considered. The inflammatory lesion presents a diffuse enlargement of the parotid without definite borders. There may

MIXED TUMORS

About 90 per cent of all salivary neoplasms are the so-called mixed tumors, and the vast majority of these affect the parotid gland. These tumors containing (1) masses of epithelial cells often showing a glandular arrangement, (2) mucoid connective tissue with mucin production, (3) cartilage, and (4) lymphoid tissue are thus truly "mixed tumors." Pathologists have long speculated as to how these tumors should be classified and disagreement still exists as to their exact place among neoplasms. The cartilage has been the major stumbling block. General opinion now is that this is really a pseudocartilage. It is believed that the cartilaginous appearance is really due to a fibrillar condensation of the myxomatous tissue with a resulting pseudocapsular arrangement around the cells.

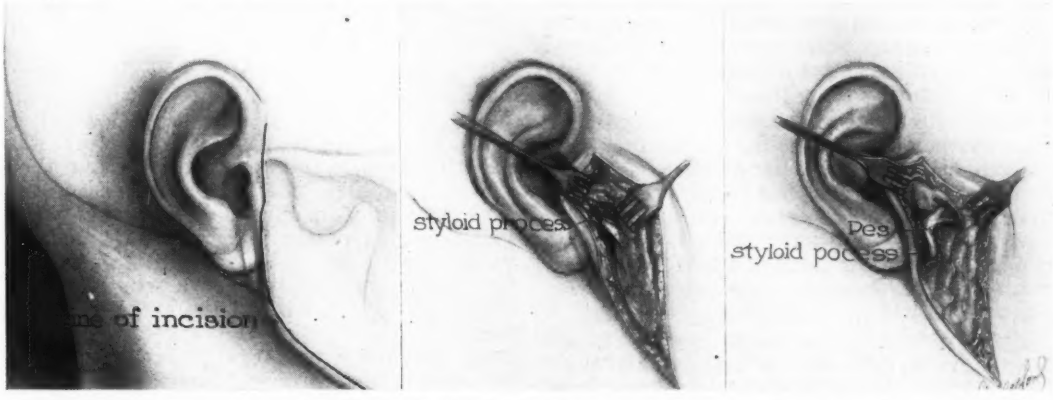

Fig. 1. Left. The usual line of incision for dissecting the facial nerve prior to removal of a parotid tumor. The dotted line shows the possible extension of the incision where the facial nerve is traced into the mastoid process.

Center. The incision carried between the capsule of the parotid gland on the right and the cartilage of the external auditory canal on the left to the base of the styloid process. The facial nerve with its accompanying vein is seen in the illustration entering the parotid gland after emerging from the medial aspect of the styloid process.

Right. Dissection of the facial nerve has been extended to the pes within the substance of the parotid gland. From here the various terminal branches of the facial nerve can readily be traced.

be tenderness of the gland or pus expressed from the duct, although this is variable. An excellent aid in the diagnosis of inflammatory lesions of the parotid gland is the presence of trismus, nearly always present. This results from the diffuse involvement of the glenoid lobe of the parotid which is pinched by the head of the condyloid process of the mandible when the mouth is opened. This sign is absent where discrete neoplasms are present.

From recent reports now available, tuberculous involvement of the parotid gland should be treated intensively with streptomycin for from four to six weeks. Actinomycotic lesions respond to a combination of penicillin and chemotherapy, although again streptomycin may be considered. Actual suppuration, of course, requires drainage and adequate cauterization.

The surgeon, however, is more interested in the clinical behavior of the cells and what kind of a prognosis the pathologist can offer. Thus the clinician attaches major importance to the history of the lesion, its physical properties, and especially its ease of removal on the operating table. This, along with the microscopic picture of the neoplasm, gives an excellent foundation on which to base the prognosis in a particular case.

At the risk of pointing out the obvious, it seems expedient to state briefly the typical picture of a mixed tumor of the parotid. The lesion is usually first noticed in early adult life as a painless swelling below and in front of the ear, just below the angle of the jaw. In older people it may have been present many years. Sudden rapid growth, beginning facial paralysis, or development of cer-

vical nodes are grave signs suggesting malignant degeneration. Because of the asymptomatic nature of the condition, some tumors reach an amazing size before medical advice is sought.

The majority of evidence today favors complete surgical excision of the parotid tumor early, although there are some authoritative sources that favor periodic observation. Dr. Carl V. Weller, pathologist at the University of Michigan, has expressed the belief that all mixed tumors are teratologic lesions (misplaced embryonal cells) which progress slowly but resolutely to a malignant degeneration. He has often stated that the fate of the patient rests with the first surgeon consulted, for unless the tumor is thoroughly and completely removed along with its capsule, the lesion will invariably recur and in a more cellular form. This recurrence may occur many years later and second and third operations are fraught with many more difficulties than the initial procedure.

Mixed tumors might well be placed in a class by themselves as is done with such neoplasms as melanomas and gliomas. Only through a vast experience has it been shown that these mixed tumors lend themselves best to surgery and that irradiation has little to offer. The latter form of therapy is usually reserved for hopeless inoperable lesions as a means of palliation only.

Embryologically, the parotid gland in its early development lies primarily lateral to the internal carotid artery, the posterior facial vein, and the facial nerve. It is especially interesting to note that the facial nerve is always medial to the principal ducts of the gland, although in its development some portions of the parotid completely envelop the nerve. Thus the surgeon can gain some solace from the fact that the facial nerve is on the deeper surface of the gland in most of its course. Occasionally a mixed tumor arises from the deeper portion of the gland, medial to the nerve, and it is here that a facial paralysis is so likely to occur unless the nerve is identified and dissected out as a preliminary procedure. Particularly in large tumors and possible malignancies of the gland, where much of the parotid gland is replaced by neoplasm, should the nerve trunk be isolated and identified.

Numerous methods of locating and dissecting the seventh nerve have been advanced. I wish to emphasize a simple and safe method that can be readily employed in every case. This method was developed and used almost routinely by Dr. A. C. Furstenberg and his associates.

An incision is made just in front of the tragus of the ear (fig. 1) from the zygoma to the angle of the jaw and if necessary along the lower border of the mandible. The incision is carried through skin and subcutaneous tissues to the cartilage of the external auditory canal. Then with a blunt elevator, the dissection is carried medialwards between parotid gland and cartilage of external auditory canal until a firm bony resistance is met. This is the base of the styloid process and is encountered at a depth of about 1.5 inches from the skin incision. The styloid process is next identified immediately below its bony base. Then with gentle traction on the parotid gland anteriorly, the facial nerve is brought into view, emerging from the medial aspect of the styloid process and curving sharply upward and laterally to enter shortly the parotid gland. The nerve is usually heavily invested in fascia and accompanied by a small artery that may prove troublesome if severed.

It is helpful in the above procedure to employ faradic stimulation in identifying the facial nerve. Once the nerve is located in this area, it can be readily traced into the parotid gland to the pes anserinus, and then as far peripherally as is expedient for the case at hand. Any surgeon doing much work in the region of the parotid should be conversant with the value of faradic stimulation in identifying the nerve trunk and its branches. A second incision can be carried postauricularly over the mastoid process from the lower part of the initial incision where it is necessary to trace the nerve into the mastoid process to gain more motility in freeing the nerve in large lesions.

There are some who advocate different methods of dealing with mixed tumors. One group, favoring watchful waiting, is merely postponing the issue at a possible risk to the patient. Slow but steady progression toward a malignant change is to be expected.

Another group favors taking a small biopsy specimen from the tumor in order to evaluate the microscopic picture. The purpose is to give the more cellular lesions irradiation, said to be beneficial on that type. The consensus, however, is that even heavy irradiation has little effect on any of the mixed tumors. A biopsy of such lesions in itself has always seemed a pointless and even dangerous procedure which could well result in seeding of tumor cells. Sound surgical principles would indicate at least a bold attempt at complete excision of such peripheral lesions.

A more recent group has advocated complete parotidectomy after first carefully identifying and freeing the facial nerve. Results as reported by Trueblood have been enthusiastic, although his series cannot be consid-

ered large enough for final evaluation. This long and tedious undertaking greatly increases the risk to the facial nerve, especially in its finer ramifications. The best argument against such a procedure is that it is probably unnecessary if the complete removal of the neoplasm can otherwise be accomplished.

Complications of operation in mixed tumors are mainly three: (1) recurrence, (2) facial paralysis, and (3) salivary fistulae. McFarland, who perhaps has collected and analyzed the largest number of mixed tumors, concludes that recurrences occur in from 25 to 30 per cent of all cases operated on for the first time. He believes that factors in recurrences are incomplete removal, seeding at the time of removal, and the possibility of a complete and separate tumor forming. He suspects this latter cause as possibly accounting for some of those recurrences after fifteen or twenty years. Again, it should be emphasized that even several recurrences of a mixed tumor should be attacked surgically as the chance for a cure is better than with irradiation alone. The fear of getting a facial paralysis may concern the surgeon more than the possibility of a recurrence and has undoubtedly been a factor in the incomplete removal of tumor in some cases.

Only occasionally is a salivary fistula troublesome as it tends to close spontaneously. In stubborn cases various plastic methods may be resorted to.

CARCINOMAS

Carcinomas of the parotid gland are not as discrete as mixed tumors. They frequently arise in a more superior part of the gland rather than in the carotid lobe (usual site for mixed tumors). The history is of shorter duration, noted in months rather than years, and there may be an associated numbness or pain. Facial paralysis is much more prone to occur in neglected cases as the carcinoma embraces the facial nerve. Prognosis of these lesions is extremely grave even in early diagnosis. Early and wide excision is essential, and this should be followed by intense irradiation delivered to skin tolerance. A block neck gland dissection is demanded by those cases presenting possible cervical metastases. Rate of recurrence for carcinoma of the parotid gland has been variously reported to be about 90 per cent.

CYSTS AND ADENOMAS

Adenomas and cysts of the parotid gland are rare. Such lesions occur usually in the upper portion of the gland, which may be a clue to their true nature. Excision of the lesion together with the capsule is essential as there is a definite trend to malignant change.

Rare tumors include lymphomas, sarcomas, and that unusual lesion, papillary cyst adenoma lymphomatosum of the parotid (Wortham's tumor). Diagnosis in such cases rests on the microscopic picture following the surgical excision of such lesions. The physician should not overlook the possibility that the parotid lesion is the site of a secondary metastasis rather than the primary lesion, hence the importance of a complete examination (including the nasopharynx) in every case.

SUMMARY

A plea is made to all clinicians to understand better the lesions of the parotid gland in order that a more uniform method of therapy may be employed. There is no need for despair in attempting to maintain the fibers of the facial nerve intact. A simple and safe method of locating and preserving the nerve trunk is described and its advantages noted. Emphasis is placed on the complete and early excision of parotid neoplasms. The fate of the patient may well rest with the first surgeon consulted.

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ABSTRACT OF DISCUSSION

Dr. A. F. Clark, Sr., San Antonio: Dr. Cody has given a concise and instructive discussion of parotid lesions. We are indeed fortunate, especially during the past five or ten years, in having modernly trained men added to the otolaryngological profession of

Texas to inspire investigation of disease conditions of this nature.

Because of the facial nerve hazard, the habit of using irradiation where surgery is indicated has become a practice. As pointed out by Dr. Cody, chemotherapy and irradiation are valuable adjuncts to surgery but do not supplant surgical drainage in suppuration of the parotid gland. Likewise, surgery is first choice in mixed tumors and malignancies of the parotid. Since the otologist and neurosurgeon have occasion to come in contact with the facial nerve problem more often than the general surgeon and since injury to the facial nerve is the big factor against parotid gland surgery, it seems only fair to the patient to have access to the specially trained surgeon.

The operation as described by Dr. Cody is ideal and sounds easy but should not be attempted unless the surgeon has been fortunate enough to have training such as is given by Dr. Furstenberg.

MANAGEMENT OF CHRONIC SUPPURATIVE TYMPANOMASTOIDITIS

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Chemotherapy and biotherapy have reduced to almost zero the incidence of acute suppurative mastoiditis, and they have lessened markedly the number of cases of acute otitis media that progress to suppuration. However, various bacterial forms develop an immunity to these therapeutic agents. This occurs when the agents are used in insufficient dosages and over insufficient periods of time in relation to the natural resistance to the drug of the particular bacterial strains involved. When immunity of the bacteria occurs in acute otitis media, the infection will become latent, the pathologic process will continue at a reduced pace, and chronic tympanomastoiditis will develop. For this reason among others chronic tympanomastoiditis will remain as an otologic problem. In coping with this problem it must be understood clearly what chronic tympanomastoiditis is, how it develops, what its variant forms are, and how to deal with these variant forms.

Chronic tympanomastoiditis may be defined as an infection of the tympanum and its adnexa in which spontaneous repair has been attempted and partially attained but has been prevented from reaching completion. If there is no attempt or an inadequate attempt at arrest and repair, the case progresses rapidly to acute suppurative mastoiditis with its complications. It is the partially successful attempt at repair that produces the chronic state, and it is the failure of consummation, or perversion, of the process that produces the various clinico-pathologic problems.

Read before the Section on Eye, Ear, Nose, and Throat, State Medical Association of Texas, Annual Session, Houston, April 28, 1948.

With one significant exception to be noted later, all cases of chronic tympanomastoiditis stem from acute suppurative otitis media, latent or manifest, rather than from acute suppurative mastoiditis. As a result of the attempted arrest and repair, varying portions of the tympanum, attic, and antrum, and usually a greater or lesser portion of the mastoid are involved in varying degrees. For this reason any treatment used must be determined by the distribution and type of lesion. Here lies the difference between acute suppurative otitis media and mastoiditis on one hand and chronic suppurative tympanomastoiditis on the other. This essential difference in the two processes and the essential difference in the therapeutic approach to each must be emphasized.

In acute suppurative mastoiditis (if no complications exist) the problem is the same in every case. It is the elimination of an acute suppurating focus. This is accomplished by total evisceration of all cells and wide opening of the antrum to obtain free drainage of the tympanum in order that normal function of the tympanum may be restored.

Each case of chronic tympanomastoiditis per contra presents a problem of its own and it must be managed according to the terms defined by this problem.

The first conditioning circumstance arises from the age of the patient at which the process begins. If the process begins before development and pneumatization of the mastoid process, no mastoid cells may develop and the entire mastoid when first observed clinically may be represented only by thick, dense cortical bone. The problem will then concern tympano-antritis alone. If the process begins after development and pneumatization of the mastoid has occurred, extensive active or healed lesions of the mastoid may be added to the tympano-antritis.

In a previous communication[3] the pathologic process occurring in the soft tissues in acute suppurative otitis media which leads to walling off and retention of isolated foci in the tympanum, the attic, and the mastoid has been described. This process is the usual one of ulceration with the reparative reactions of proliferation and fibrosis. However, aided and abetted by the particular structural characteristics of the tympanum, attic, and antrum, these reparative processes never reach consummation and suppurating foci remain. Such foci may continue their ulcerative and erosive course leading to complete destruction of the tympanic membrane and the tympanic mucosa, and necrosis of the bony walls of the tympanum and adnexa.

On the other hand the reparative process may be sufficient to limit the process to the attic and aditus alone, allowing both the tympanum and the mastoid to escape serious involvement. Here it should be noted that it is doubtful if the attic ever escapes involvement in this disease. I have seen cases in which a perforation of Shrapnel's membrane has led to surgery only to find that the tympanum has remained essentially normal with extensive pathologic disturbance in the attic. Never have I found chronic suppuration in the tympanum without involvement of the attic. The anatomy of the tympanum and its attic and the pathologic processes already described[3] account for this observation.

In addition to proliferation and fibrosis a third reparative process must be reckoned with. This is stratified squamous epithelial invasion. If at any point in the tympanum normal squamous epithelium arising from the external auditory canal or the outer layer of the tympanic membrane is brought into contact with an ulcerative process of the tympanic mucosa which is attempting repair, the epithelium joins in this process by invading and attempting to overlay the ulcerating area. If this epithelial invasion occurs into a pocket with a narrow outlet from which the naturally desquamating layers of epithelium cannot escape, it leads to the formation of a cholesteatoma.

At this point may I correct a commonly accepted fallacy. A perforation does not have to be marginal to produce a cholesteatoma. I have found cholesteatomata at operation in cases with only central perforations.

Here too may I refer to the previously mentioned exception to the rule that all cases of chronic suppurative tympanomastoiditis succeed acute suppurative otitis media. During the resorption of the embryonic mesenchymal tissue in the middle ear and attic during the first year of life Shrapnel's membrane in rare cases may be pulled inward by the retreating mesenchyme so that it forms a deep pocket.[6] The squamous epthelium of this membrane forming the lining of such a pocket will be incarcerated as it desquamates. The successively desquamated layers will form a cholesteatoma which will erode by expansive pressure the anterior and posterior walls of the notch of Rivinus and the outer wall of the attic. This erosive process will continue in all directions until the entire attic and antrum are involved. If infection of this mass occurs via the external auditory canal, as it almost always does eventually, there will be the same problems as are caused by a cholesteatoma formed during the course of chronic tympanomastoiditis of orthodox origin.

TREATMENT

With these facts clearly in mind the problems of treatment may be approached. Not every case is a case for surgery and medical treatment may be the method of choice. There are definite indications for each and these indications should govern the procedure. When surgery is called for it should not be delayed.

Medical treatment may be the method of choice when the following conditions are met: There must be (1) no active process in a pneumatized mastoid, (2) no incarcerated cholesteatoma, (3) no progressive necrosis in the tympanum or attic, and (4) no evidence of involvement of the internal ear, the facial nerve, or the meninges.

When a case is being treated medically, all aqueous solutions to be instilled by the patient and all forms of aqueous lavage must be avoided. In addition to propagating the infection to hitherto uninfected areas, such modalities may serve to increase the swelling and expansive pressure of a cholesteatoma.

By the use of attic canulas, which may be fashioned quite easily from 16 gauge spinal needles after grinding away the point and bending to the desired shape after heating, the attic and tympanum may be lavaged with 70 per cent alcohol for cleansing purposes. Profuse granulations that are accessible may be cauterized or removed with biting forceps and iodine powder or the sulfonamides insufflated. Infection or occlusion of the eustachian tube should be dealt with by the use of local medication and nasopharyngeal radium application. In spite of such treatment a moderate discharge may persist. This alone should not be cause for abandonment of medical treatment and the institution of surgery, nor should promises be made to the patient that surgery will cause it to disappear.

However, surgery should be advised and even urged when any of the following factors are present: (1) an active process in the mastoid, (2) an incarcerated cholesteatoma, (3) evidence of progressive necrosis in the tympanum or attic, (4) evidence of the extension of the process to the internal ear, the facial nerve, or the intracranial cavity, and (5) evidence of increased sepsis.

In considering surgery for the relief of chronic tympanomastoiditis, all concepts of the radical or modified radical tympanomastoidectomy being a further step of the so-

called "simple" mastoidectomy must be forgotten. The early European otologists, notably Stacke[4] and Politzer,[2] recognized the essential difference, but a majority of the English and American otologists from Whiting[5] to the most recent authors of texts on otology start the description of the operation with some variation of the statement, "Complete a simple mastoidectomy and then proceed as follows." This is an error productive of further errors with ultimate failure of the operative effort. The operation must be approached mentally and technically as an entity totally separate and apart from simple mastoidectomy. This operation has for its purpose the completion of an aborted or perverted process of repair having its start, and in the majority of cases its principal focus, in the attic and antrum. It is *at* this point that surgery should begin and it is *from* this point that it should progress in all directions until the work has been completed.

Before any operative step is taken it must be decided if the entire tense portion of the tympanic membrane and the tympanum may be preserved by the performance of a modified radical tympanomastoidectomy or if the entire tympanum and tympanic membrane must be sacrificed by the performance of a total radical tympanomastoidectomy.

SURGICAL TECHNIQUE

There are now available two approaches and two techniques for this surgery: the postauricular approach with chisel and gouge technique and the endaural approach with the technique of the electrically driven burr, the long handled curets, and the fine jawed rongeurs. With either method the objectives are the same.

There must be adequate exposure of the field of bone surgery. The tympanomastoid suture must be identified as soon as the soft tissues are elevated, for in the postauricular route it defines the position of the facial nerve, and in the endaural route it defines the fibrous attachment of the cartilaginous external auditory canal.

Surgery of the bone in the postauricular route should be started by placing the gouge at the postsuperior angle of the external opening of the bony portion of the external auditory canal and chiselling inward toward the middle ear, removing the bone in thin layers. This procedure is continued until the postsuperior canal wall and the outer wall of the attic are removed. Bone removal continues so that when the first stage is completed the bridge will have been removed and the cavity created in the bone will be kidney shaped, the poles of the kidney lying respectively over the periantral region and the anterior epitympanic space, and the pelvis of the kidney over the attic.

If the endaural approach is used and if a modified tympanomastoidectomy is projected, the perforating drill should enter the fossa mastoidea and it should be directed inward parallel to the roof and posterior wall of the external canal. By this route the antrum is entered immediately. Round burrs are then used to develop the kidney shaped bony wound, the cutting being always outward from the antrum and away from all danger sites. If total tympanomastoidectomy is contemplated, a small round cutting burr is introduced within the external canal and engaged directly under the outer wall of the attic as the initial step. The cutting is then directed outward and the kidney shaped wound is developed. In either method all diseased tissue, bony and soft, must be removed.

If either modified or total tympanomastoidectomy is being performed, the bridge with its postero-inferior and antero-superior abutments must be removed completely.

In total tympanomastoidectomy the eustachian tube proper should not be curetted although all peritubal cells must be removed. If the canal for the tensor tympani muscle is eroded, the entire muscle must be removed. Suppuration of this muscle is confused frequently with postoperative tubal suppuration. The floor of the external auditory canal must be removed to the lowest level of the hypotympanum and all remnants of the tympanic membrane and its annular attachments must be removed.

Any cholesteatoma present must be removed completely. If the matrix is smooth and free of any ulceration, it may be left where it overlies bone as it represents just that much epithelialization accomplished. If there is any ulceration of the matrix, the matrix must be removed. If there is the slightest doubt of the presence or absence of ulceration, the matrix must be removed.

If the postauricular route has been used, the final step of the operative procedure is the formation of the plastic flap. In my hands the Daily flap[1] has proved to be best. If the endaural route has been used the flap will have been made early in the operation, and the triangular section of the skin removed as the initial incisions were made may now be replaced as a half-thickness graft. This graft should be placed where most needed as determined by the final shape and size of the bone cavity.

It is my custom to pack the wound with Carbozine gauze and to remove this pack on the fifth postoperative day. No pack is replaced but the wound is watched carefully

and all clots and fibrin masses are removed promptly and all exuberant granulation tissue is bitten away under local anesthesia.

For the past two years I have used the endaural method exclusively. I have found no case in which it has not proved superior to the postauricular approach. Nor have I found any case which, because of the extent of pneumatization or because of complications, could not be completed by this method. Its specific advantages may be enumerated as follows: (1) It provides ample exposure for the surgical approach to all portions of the field. (2) It provides the most direct approach both visually and surgically to all portions of the field. (3) It provides immediate exposure of the antrum and attic so that all subsequent procedures are directed away from dangerous areas. (4) The initial incisions delineate the flap so that one of the most tedious tasks is avoided at the end of the operation. (5) In the modified tympanomastoidectomy it provides much greater safety and ease in the formation of the plastic flap in continuity with, and for the preservation of, the tense portion of the tympanic membrane.

CONCLUSIONS

1. Because in the future chronic tympanomastoiditis will be the infectious disease of the temporal bone most frequently encountered, ability to perform the total or modified tympanomastoidectomy must be brought to its best technical development.

2. Chronic suppurative tympanomastoiditis poses entirely different surgical problems than does acute suppurative mastoiditis.

3. For this reason the surgical correction of this disease must be mentally and technically divorced from that of acute suppurative mastoiditis.

4. For this purpose there are now two surgical techniques with the same objectives.

5. In my hands the technique of the endaural approach has proved to be the more satisfactory.

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ABSTRACT OF DISCUSSION

Dr. John H. Barrett, Houston: It is extremely difficult to open the discussion of a paper that has so completely covered the subject as Dr. Sellers has in his presentation. The term chronic suppurative tympanomastoiditis is more descriptive of the pathologic process than any of the other titles for this disease. Many terms in otology need a revision. The terms "simple radical" and "modified radical mastoidectomy" are misleading to both the student and physician. A simple mastoidectomy is far from being simple and in most cases the pathologic disturbance is more extensive than in the radical mastoidectomy when the disease process is often limited to the tympanum and mastoid antrum. The term "complete mastoidectomy" should be the name of this operation and the term "radical mastoidectomy" should be changed to "tympanomastoidectomy." The modified radical mastoidectomy is really a modified or better a conservative tympanomastoidectomy.

The alteration of middle ear and mastoid disease by chemotherapy and biotherapy is becoming a more confusing and baffling problem. In addition to the infections caused by sulfa and penicillin resistant organisms there are some infections in which the virulence is reduced but the disease process is not entirely cured. Cases of chronic catarrhal otitis media have developed in the presence of these therapeutic agents. These cases also show lymphoid hyperplasia in the region of the eustachian orifices and roentgen study of the mastoid usually reveals some mastoiditis. Treatment of the eustachian orifices by radium application has been beneficial in many cases. In certain cases in which the condition has persisted roentgen-ray treatments over the mastoid help. It will be interesting to examine patients fifteen or twenty years from now and see if there is any conductive deafness and undeveloped mastoids in the cases of acute middle ear and mastoid infections which are now being cured by chemotherapy and penicillin.

I agree with Dr. Sellers in his indications for surgery. In such cases I attempt to explain the disease process and possible complications to the patient and then advise him that I recommend surgery and am prepared to do it whenever he is ready. I prefer the endaural approach for a tympanomastoidectomy except where other postauricular surgery has preceded the operation. I believe that magnification with a binocular loupe should be employed in cleaning the structures of the tympanum. Most failures in the so-called radical mastoidectomy have been due to improper and incomplete removal of infected tissue from the middle ear cavity. Of equal importance is the removal of an infected tensor tympani muscle. Contracture of the external meatus is a complication of the endaural approach but can be avoided in making the incisions either in the Lempert technique or the Shambaugh modification and removal of the underlying tissue and periosteum will reduce the occurrence.

FOUR TYPES OF SURGERY ABOUND

Appendectomies, tonsillectomies, obstetric, and gynecologic procedures account for approximately 60 cents out of every $1 paid to physicians by Blue Shield Plans for surgical service, according to Associated Medical Care Plans. Beyond these major cost items, approximately seventy surgical procedures will absorb 86 per cent of all payments to physicians for benefits provided under the average surgical type of subscriber contract.

Standard reporting forms are being completed by the Blue Shield national office, with the exception that complete actuarial data for 1948 will be reported and tabulated by the middle of 1949, thus fulfilling one of the major functions assigned to Associated Medical Care Plans at the time of its incorporation in 1946.

THE ROLE OF CALCIUM IN BONE DISORDERS

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Present knowledge of bone disorders is extremely incomplete, due in great part to a lack of understanding of the anatomy and pathology of bone itself. Bone cannot be studied until decalcified; as a result much valuable information is lost. Progress is now being made largely from a physico-chemical approach, namely, calcium metabolism. As a result of these studies, the numerous disorders once classified only on a roentgenologic basis may now be divided according to etiology and mechanism of production.

CALCIUM METABOLISM

A discussion of the theories and relationships of normal calcium metabolism is a requirement for the understanding of these mechanisms. They may be divided into four main categories, each closely related to and dependent upon the others.

Calcium Absorption.—For this process a number of prerequisites must be fulfilled:

1. Adequate intake. On the average from 0.65 to 0.7 Gm. daily, though it may equal from 1 to 2 Gm. for pregnant women or growing children.
2. Optimal intestinal hydrogen ion concentration. Calcium tends to be precipitated in an alkaline medium.
3. A diet of not more than 10 per cent fat which must be digested completely; bile salts are an important adjunct.
4. A correct proportion of other minerals. The ratio of calcium to phosphates should be at least 2 to 1. Sodium, potassium, lead, and magnesium may replace calcium; aluminum and ferric iron may free it for absorption.
5. Amino acids are thought by McCance and others to increase calcium solubility and absorption.
6. Vitamin D in normal therapeutic doses facilitates calcium absorption.

Blood Calcium.—This category concerns ionizable calcium, which constitutes 55 per cent of total blood calcium or from 5 to 6.5 mg. per 100 cc. of blood. It is affected by:

1. Calcium absorption, which has already been discussed.
2. Parathyroid gland, which has an indirect effect. The mechanisms will be discussed later.
3. The acid-base balance. Solubility of calcium is increased in acidosis and decreased in alkalosis.
4. Serum phosphate, which has a reciprocal relationship. The other elements mentioned under absorption may in addition affect blood calcium.
5. Hypervitaminosis D produces hypercalcemia, the calcium being mobilized from the bones.
6. The remaining 45 per cent of total blood calcium or from 4 to 5 mg. per 100 cc. of blood is serum calcium, bound to plasma proteins and thus nondiffusible.

Calcium Excretion.—Seventy-five per cent of this is fecal, representing a constant endogenous amount plus unabsorbed calcium. The remaining 25 per cent is renal and affected by:

1. Serum calcium level as discussed.
2. Other elements, as already discussed, may also affect calcium excretion.
3. Parathyroid. One of the primary results of hyperparathyroidism is a lowering of the renal threshold for phosphates. This increases their excretion with consequent mobilization of calcium phosphate from bone, producing hypercalcemia and hypercalcinuria.
4. Thyroid. In contradistinction to Aub's theory, Robertson has recently shown that in hyperthyroidism the renal threshold for calcium is lowered.

Calcium Metabolism in Bone.—This is not a static process; calcification and decalcification are going on continuously and simultaneously throughout life. The exact mechanism of calcification is believed to be that advanced by Robinson, that is, the hydrolysis of phosphoric esters by an enzyme, phosphatase, produces a local increase in phosphate ions which, if sufficient calcium be present, causes precipitation of calcium phosphate. Calcification may occur directly without this enzyme action if the concentrations of calcium and phosphates are sufficiently high. Requirements in general for normal calcification are (1) a suitable organic matrix with a relatively high hydrogen ion concentration and containing phosphatase and (2) an adequate blood supply. Vitamin D is thought to have some specific effect on this phase of calcium metabolism, and vitamin C is believed to affect the formation or stability of bone phosphatase. Estrogens are known to produce hyperossification when given experimentally, and Selye has demonstrated a direct parathyroid effect on bone itself.

BONE DISORDERS

With this background a consideration of the mechanisms of production and the principles of treatment of the various bone disorders may be undertaken. Symptomatic therapeutic measures will not be discussed.

Read before the Section on Radiology and Physiotherapy, State Medical Association of Texas, Annual Session, Dallas, May 6, 1947.

For comparison, schematic diagrams of calcium metabolism in the most frequently encountered malacias as originated by Dr. Fuller Albright are included. Figure 1a represents the normal.

Senile Osteoporosis usually occurs in women beyond the menopause and particularly involves the spine and pelvis although it is a generalized process. It is thought to be due to a decreased osteogenic activity of the osteoblasts without a parallel retardation of decalcification. Albright separates women under 65 years of age into a premenopausal and a postmenopausal group, in the latter of which he believes the decreased osteoblastic activity is the result of estrogen withdrawal. However, such an etiology does not explain its appearance after the age of 65, nor its occurrence in males since androgens apparently do not have the same effects on bone. The calcium metabolism is shown in figure 1b. Serum calcium and phosphates and blood alkaline phosphatase are all within normal limits, as are calcium and phosphate excretion values. Regarding therapy, little can be expected from calcium, phosphates, and vitamin D themselves, although intake should be adequate. Stilbesterol has produced definite improvement in a fair proportion of women under 65, but its use is not without dangers. Aluminum acetate has been used empirically with subjective relief in many cases and occasional objective signs of improvement; its means of doing so is unknown.

Osteomalacia is seen rather infrequently in this country. The underlying mechanism is a failure of calcification of the osseous matrix due to a lack of availability of calcium. The etiology may be insufficient calcium intake as in repeated steatorrhea or sprue, or may be a deficiency of vitamin D. Calcium metabolism is outlined in figure 1c. Serum calcium is normal or slightly decreased, serum phosphates usually decreased, and blood alkaline phosphatase generally increased. There is a decreased excretion of calcium and phosphates. Therapy consists of calcium, phosphates, and vitamin D by oral and parenteral administration. Liu and Chu reported excellent results with the substitution of dihydrotachysterol for vitamin D and with the use of fer-

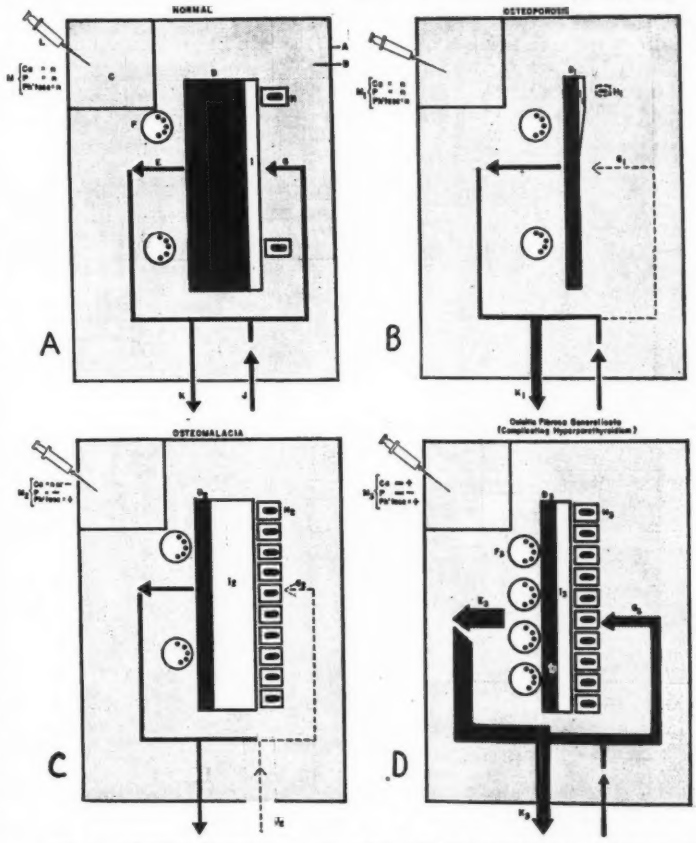

FIG. 1. Schematic diagrams by Dr. Fuller Albright showing the differences in normal bone metabolism, osteoporosis, osteomalacia, and osteitis fibrosa generalisata complicating hyperparathyroidism.[After Albright, Smith, and Richardson: Postmenopausal Osteoporosis, J.A.M.A. 116:2465-2474 (May 31) 1941.] *A*, body limits; *B*, body fluid; *C*, body serum, a compartment of body fluid easy to tap for analysis; *D*, bone mass with two surfaces, one at which bone is being resorbed and one at which it is being laid down; *E*, arrow indicating by its size the rate of calcium and phosphorus resorption; *F*, osteoclast; *G*, rate of calcium and phosphorus deposition; *H*, osteoblast laying down osteoid (*I*); *J*, calcium and phosphorus entering the body from the gastro-intestinal tract; *K*, calcium and phosphorus leaving the body by the kidneys and other exits; *L*, syringe for obtaining serum for analysis; *M*, blood values (n, normal; +, high; and —, low).

a. *Normal.* Note that the deposition of calcium and phosphorus (*G*) equals the resorption (*E*) and that part of the latter goes back into the bone.

b. *Osteoporosis.* Note the decrease in bone mass (D_1), the primary hyperplasia of osteoblasts (H_1), the decreased deposition of osteoid (I_1), the decreased deposition of calcium and phosphorus (G_1), the increased excretion of calcium and phosphorus (K_1), and the normal blood values (M_1).

c. *Osteomalacia.* Note the decrease in bone mass (D_2), the hyperplasia of osteoblasts (H_2) because of increased stresses and strains, the increased deposition of osteoid (I_2) inadequately calcified because of the low serum calcium and phosphorus values, the decreased deposition of calcium and phosphorus (G_2), the primary difficulty in absorbing calcium and phosphorus from the gastro-intestinal tract (J_2), and the abnormal blood values (M_2).

d. *Osteitis Fibrosa Generalisata Complicating Hyperparathyroidism.* Note the excretion of calcium and phosphorus in the urine (K_3), the increased resorption of calcium and phosphorus (E_3), the increase of osteoclasts (F_3), the decreased bone mass (D_3), the increased bone formation by osteoblasts (I_3) because of increased stresses and strains, the increased deposition of calcium and phosphorus (G_3) because the serum is not undersaturated in respect to calcium phosphate, and the high phosphatase level (M_3).

ric iron; the latter combines with negative ions in the gastro-intestinal tract, thus releasing free calcium ions for absorption.

Hyperparathyroidism generally occurs in young to middle-aged women. In the past ten to fifteen years realization of its occurrence without bone changes has brought about a marked increase in diagnosis. The parathyroid pathology may be either an adenoma or a generalized hyperplasia which is usually of the Wasserhelle type. Figure 1d schematically represents calcium metabolism. The means of action is thought to be a lowering of the renal threshold for phosphates, thus depleting blood phosphates. Mobilization of phosphates from the bone as calcium phosphate will thus increase serum calcium and urinary calcium. There is present hypercalcemia, hypercalcinuria, hypophosphatemia, and hyperphosphaturia. Blood alkaline phosphatase is increased. If there is an adequate intake and absorption of calcium and phosphates to maintain normal blood levels, the bones are unaffected. This is now most frequently the case. Therapy consists of removal of the adenoma or of most of the hyperplastic parathyroid tissue. Calcium, phosphates, and vitamin D are given in addition.

Hyperthyroidism, according to the recent work of Robertson, involves the mechanism of lowering the renal threshold for calcium itself. This increases the calcium requirement directly, which may be satisfied by adequate intake and absorption or may deplete the bony storehouse. Serum calcium is normal or decreased, serum phosphates are normal or increased and blood alkaline phosphatase is increased. Hypercalcinuria and hyperphosphaturia are found. Ordinarily hyperthyroidism must be present over a period of at least five years to produce noticeable osteoporosis. Therapy is directed at the gland itself, with administration of calcium, phosphates, and vitamin D as useful adjuncts.

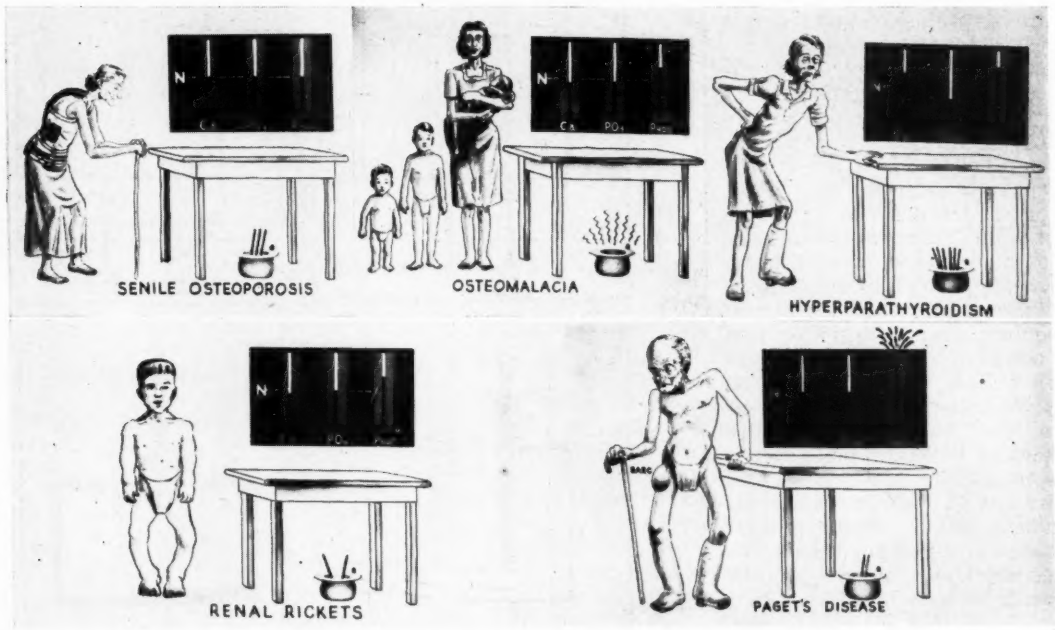

FIG. 2. Diagrammatic representations of the usual characteristics of several bone disorders:
Senile osteoporosis—elderly woman, back pain and deformity, blood chemistry, urinary calcium.
Osteomalacia—may be undernourished female with frequent pregnancies or may be foul-smelling stools of steatorrhea, blood chemistry.
Hyperparathyroidism—relatively young asthenic woman, renal colic, tendency to fractures, blood chemistry, urinary calcium.
Renal rickets—young child, bow legs, thick ankles and wrists, short thick hands and feet, square skull, blood chemistry, urinary calcium.
Paget's disease—elderly male, large head, bowed femora and thick bowed tibiae, tendency to secondary osteogenic sarcoma, blood chemistry, urinary calcium.

Renal Rickets is the outstanding malacia seen in children. There are varied mechanisms and pathology producing this picture, although the underlying etiology is chronic renal insufficiency. Acidosis plays a part in these cases. In addition, phosphate retention reduces blood calcium, and increased phosphate excretion into the bowel decreases calcium absorption. Thus calcium from the bones is utilized. Serum calcium is usually normal, serum phosphates are elevated, and blood alkaline phosphatase is increased.

Therapy is a difficult problem since the chronic renal insufficiency is intractable. Generally recommended is a diet high in calcium and low in phosphates. Vitamin D has been used in addition, though Liu and Chu have recommended dihydrotachysterol (AT-10) instead. They suggested also the administration of ferric iron to combine with phosphates and other negative ions forming insoluble compounds and thus to increase free calcium ions for absorption.

Osteitis Deformans, originally described by Sir James Paget in 1876, has an unknown etiology. Presently advanced theories include (1) a local destructive process followed by compensatory new bone formation and (2) exhaustion hypoparathyroidism. The entity involves men beyond middle age predominantly and is noted for its chronicity. Recently the number of monostotic cases have increased, probably because of increased use of roentgen ray as well as better recognition. Blood and urinary findings as regards calcium and phosphates are within normal limits. Blood alkaline phosphatase is elevated and frequently markedly so, its most extreme values being seen in Paget's disease. Therapy has brought about indifferent results. Calcium, phosphates, and vitamin D seem to have been of little value, and they brought extensive metastatic calcification in 1 case reported by Wells and Holley. Reifenstein and Albright have pointed out a similar danger following prolonged immobilization. Roentgen therapy has been used to control pain in the few cases requiring it. Vaccines of various types have been advanced from time to time. Recently, aluminum acetate has been used empirically with subjective improvement.

There is a constant addition to the list of bone malacias. The osteosclerosis of fluorine etiology, seen in cryolite workers or in persons drinking water containing greater than 1 part per million of fluorine, is now well recognized. And physicians are becoming aware of the osteoporosis seen in approximately 2 per cent of hypertensive patients under potassium thiocyanate therapy, probably the most recent addition to the group and for which attempts are at present being made to work out the mechanism of production.

However, understanding of these probems is far from complete. Most of the theories of calcium metabolism, normal and abnormal, are unproved. Therapy is specific in only a few of these disorders and produces indifferent results in the others. And there still remains a group, such as osteogenesis imperfecta and osteopetrosis, about which nothing of the underlying metabolism or pathology is known. Much progress has been made in the understanding of bone from a physicochemical standpoint; much more remains in the offing.

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ABSTRACT OF DISCUSSION

Dr. Ben DuBilier, Austin: Any patient presenting the roentgenographic evidence of multiple cystic lesions or even a localized cystic lesion should be subjected to calcium phosphorus and phosphatase studies. Dr. Hinchey has stressed the importance of calcium determinations. Although he has not placed as much emphasis on the phosphorus determination he has stated that there is an important reciprocal relationship between calcium and phosphorus levels. Only when there is kidney impairment does this relationship change and this is an important feature of secondary hyperparathyroidism. Important bearings upon treatment and prognosis will ensue from this differentiation.

In hyperthyroidism there is an increased excretion, not only of calcium but also of protein. Dr. Hinchey has mentioned the intimate relationship of nondiffusible calcium and protein. Is it possible that the protein excretion is really the cause for the calcium excretion?

Another point of interest relating to the protein-calcium ratio is that in some hypocalcemic states, if a correction were made for the low serum-protein, a true hypercalcemia might exist.

The underlying mechanism of osteomalacia, as suggested, is a failure of calcification of the osseous matrix. Could this mechanism rather be a washing out of calcium from a healthy matrix, dependent on a lack of vitamin D? These patients make good response to vitamin D therapy.

The detoxifying efforts of the bone, particularly to lead, radium, arsenic, and fluorine, should be kept in mind.

We are grateful to Dr. Hinchey for his analysis of the physico-chemical status of calcium metabolism. A careful analysis of calcium in relation to phosphorous is a definite aid to the radiologist in evaluating osteoporotic and cystic bone lesions.

CHRONIC INFLAMMATORY LESIONS OF BONE RESEMBLING NEOPLASMS

REPORT OF 3 CASES

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It is sometimes astounding how much an inflammatory lesion can resemble a bone tumor and the authors wish to report 3 cases in which the differentiation was impossible clinically and radiologically. In 2 of these cases a tumor was believed to be the most likely diagnosis and an inflammatory process was considered in the differential diagnosis. In the third case a neoplasm was the only diagnosis seriously considered.

With chemotherapy using the sulfonamides and penicillin, cases of aborted acute hematogenous osteomyelitis frequently develop roentgen-ray changes closely simulating bone tumors, and the surgeon must constantly be on guard to keep from making a diagnostic error and to keep from overlooking a developing tumor. None of the 3 cases being reported, however, had received any chemotherapy before being subjected to biopsy. None of the cases ever suppurated, and yet they all were inflammatory in nature, being periostitis or osteoperiostitis.

While sclerosing nonsuppurative lesions about the long bones are not too uncommon, there is a type of case resembling a bone tumor wherein there is not only the sclerosing feature of the periosteum, but the appearance of the medullary cavity also leads the physician to suspect a more malignant lesion than actually exists. This type of lesion is not frequently described in the literature.

It is true these cases at first glance appear to be related to the nonsuppurative periostitis of Garre. However, careful evaluation of the differential elements that go to make up the latter will show a marked difference. Also, they might be suspected to be cases of chronic osteomyelitic processes with areas of lesser density in the shaft that simulate cavities. There is a marked difference from them, however.

While each of the 3 cases presented is dissimilar in roentgen-ray appearance, they are similar histopathologically. They seem to fall into no particular classification of the periostitides as currently described in texts and literature on pathology. They were a nonsuppurative, nonspecific inflammation of bone. The cases are presented for their interest as diagnostic problems. Each case has been followed for over a year following the biopsy and has had a cure clinically with relief of symptoms.

Read before the Section on Clinical Pathology, State Medical Association of Texas, Annual Session, Houston, April 27, 1948.

CASE REPORTS

CASE 1.—E. F., a 13 year old Mexican boy, was first seen on May 6, 1946. He complained of pain in his left leg for three months; and pain with swelling one month prior to being seen. His pain was worse at night, was constant, and extended from the tibial tubercle to the dorsum of the foot.

On physical examination there was tenderness and swelling over the upper and middle one-third of the left tibia. The child appeared undernourished, and his mother stated he had lost 10 pounds of weight in the past month. There were scattered rales throughout the chest. Considerable mucopurulent postnasal discharge was present. His temperature was normal. There were no other significant findings.

Laboratory findings were as follows: The leukocyte count was 8,900, with 71 per cent polymorphonuclear cells. The red blood cells and hemoglobin were normal. The urine examination was not significant. Repeated Kahn tests were negative. The typhoid agglutination was positive in a titer of 1:80.

Roentgenographic examinations of the left tibia revealed a dense fusiform enlargement of the cortex of the upper third of the left tibia without evidence of destruction or reaction. In the chest roentgenogram there was extensive bronchial thickening in both lungs, with a few calcified nodes in the hilium areas, but without evidence of any activity of the lesions.

On May 20 the area of the tumor was explored. There was thickening and proliferation of the periosteum. The bone was soft on cutting it. A specimen of pinkish-gray, firm, cancellous and cortical bone 8 mm. in diameter was removed with the periosteum. Microscopic examination revealed no changes in cancellous or cortical bone. There was marked fibrocytic hyperplasia of the periosteum with eosinophylic myelocytes, leukocytes, plasma cells, and hyperchromic fibrocytes. There were many newly formed vessels lined by swollen endothelial cells. There was no evidence of new bone formation or neoplasia. Microscopically, the diagnosis was fibrosis and granulation.

The operative wound healed without infection. There was relief of pain within one week of operation. The patient was discharged to return at intervals for follow up. On November 4 roentgenographic examination revealed regression of the fusiform enlargement and only slight spindling remained. The patient remained symptom free and at the time of his last examination over a year after the original onset of his symptoms he had no complaints and the leg was normal on examination.

The differential diagnosis between this case and Ewing's tumor was most difficult to make as far as the roentgenogram and the clinical appearance of the patient were concerned. A biopsy was really the only way to make an accurate differential diagnosis. Other things which should be considered in the differential diagnosis in this case are osteogenic sarcoma of the sclerosing type involving chiefly the periosteum, typhoid infection of the bone, and syphilis.

CASE 2.—P. C., a 58 year old man, was first seen on April 16, 1946. He complained of a painful right foot. He had had pain for one month. Questioning as to other episodes revealed that he had had painful arches off and on for years.

Physical examination revealed tenderness and pain on standing about the longitudinal and metatarsal arch of the right foot. The longitudinal arch was moderately depressed.

A support with a longitudinal and metatarsal component was prescribed. The patient became slowly worse, developing pain over the right third metatarsal bone and slight swelling of the dorsum of the foot.

Roentgenographic examination June 13 revealed an unusual lesion of the anterior one-third of the right third metatarsal bone. There was an area of cortical bone destruction, with periosteal reaction, and involvement of the medullary canal as well. The picture was not characteristic of either march fracture, inflammation, or neoplasm. The chest revealed no pathologic process. Laboratory examination of the blood and urine was not significant and the Kahn test was negative.

On July 18 a biopsy was done, along with a partial excision of the lesion. There was a fusiform, infiltrative, discrete process about the proximal one-third of the metatarsal shaft involving the cortex. The periosteum was thick. The gross appearance was that of a granulating process. Microscopic examination of the tissue revealed compact hyalinized fibrous tissue with focal areas of lymphocytic infiltration. Thick walled vessels were prominent. Examination after decalcification of the bone showed small islands of necrosis. Other areas showed fibrocytic hyperplasia with minimal leukocytic infiltration. There was no evidence of neoplasia. The microscopic diagnosis was chronic inflammation.

The operative wound healed uneventfully. The patient did well following the operation and after about six months was nearly relieved of his pain. At the end of a year he still had a litttle chronic low grade pain in his arches but was not disabled. The last roentgenogram made six months after operation showed good repair of the bony lesion.

In the differential diagnosis in this case an osteogenic sarcoma would be the most likely diagnosis. In this particular case, an inflammatory lesion was considered a likely possibility and a march fracture was also seriously considered as a diagnosis. There is a remote possibility that this case could be a march fracture but radiologically there was too much erosion and pitting of the bone and microscopically the changes were not those to be expected. A mixed osteolytic, osteoplastic type of osteogenic sarcoma would give a roentgen-ray picture much like this one. Other things to be considered in the differential diagnosis of this case would be metastatic carcinoma and some other type of inflammatory process in the bone.

FIG. 1. CASE 1. A chronic nonsuppurating inflammatory lesion of bone closely resemble a Ewing's endothelial myeloma in a 13 year old boy. No suppuration ever occurred.

FIG. 2. CASE 2. A chronic nonsuppurative lesion of a metatarsal bone in a 58 year old man. No suppuration ever occurred.

FIG. 3. CASE 3. A chronic nonsuppurative destructive process of the distal fibular epiphysis in a 3 year old boy. This lesion closely resembled an osteolytic osteogenic sarcoma. No suppuration ever occurred.

CASE 3.—J. E. W., a 3 year old white boy, was first seen on April 11, 1946. The mother stated the child had had a tender, painful left ankle since a twisting injury on February 6.

On physical examination there was tenderness and moderate swelling about the external malleolus of the left ankle joint. The child limped and resisted efforts to move the ankle through a full range of motion. The temperature was normal and his general condition was good. Laboratory examinations of the

blood and urine were not significant. The Kahn test was negative.

Roentgenographic examination revealed an irregular destructive process without evidence of bony reaction, involving the distal fibular epiphyseal line, the tip end of the fibula, and distal portion of the metaphysis. There was moderate osteoporosis. The chest roentgenogram revealed no pathologic process.

On April 12 a biopsy of the region was done. A tumor-like substance was found, discrete, noninvasive, white, and of a cartilaginous consistency. Microscopic examination of several sections showed cartilage of an adult pattern, with the cells uniform and in orderly alignment. The periosteum was thickened, accompanied by increased vascularity. The larger vessels showed thickening of the adventitia; the smaller vessels showed swelling of endothelial cells. There was leukocytic infiltration with small lymphocytes. The process was of endarteritis and chronic periostitis, without evidence of neoplasia in any of numerous sections studied.

The postoperative course was uneventful. The foot was immobilized in a short leg plaster cast and the symptoms soon disappeared. Subsequent check-ups found the patient to be asymptomatic. Roentgenographic examination on December 18 showed good repair of the destructive process with considerable irregularity still present.

Reexamination clinically at the end of one year revealed a normal ankle; the patient had no symptoms and he walked without a limp.

This particular case was considered to be in all probability an osteogenic sarcoma. With the involvement primarily in the region of the epiphyseal line and with a good deal of involvement of the epiphysis, the primary diagnosis of an inflammatory lesion was not considered likely by the authors although this is what it turned out to be. An osteolytic destructive process of this type seemed to be most likely due to an osteogenic sarcoma. A Ewing's tumor was considered as a second possibility. Even at the time of operation the gross appearance of the tissue was suggestive of an osteogenic sarcoma and it was not until a careful study of the material was done that neoplasm could be entirely ruled out.

DISCUSSION

The radiologic and clinical diagnosis, as well as the histopathologic diagnosis of bone lesions remains one of the most difficult of diagnostic problems in spite of the excellent work of the Bone Tumor Registry. The correlation and evaluation of all available information is essential and requires the full cooperation of the surgeon, radiologist, and pathologist.

The use of sulfonamides, penicillin, and streptomycin has complicated the problem of differential diagnosis not only for the radiologist but for the pathologist, and we may expect to see more unusual and atypical cases with the continued use of chemotherapy.

The cases presented in this paper demonstrate clearly that no matter how sure the physician may feel that he is correct in a diagnosis of a neoplasm, radical treatment should never be instituted without a biopsy in which an adequate amount of tissue is removed and carefully studied by a competent pathologist. Small fragments of soft, structureless, grumous material are not satisfactory for study and the biopsy should be large enough to include firm periosteal tissue, cortical bone, and medullary structure if possible.

The differential diagnosis in the 3 cases presented was extremely difficult and the diagnosis of neoplasm was favored over an inflammatory process until the biopsy proved the true nature of the lesion. In addition to the malignant lesions that are frequently mistaken for an inflammatory process other conditions that are confusing include: osteoid-osteoma; monostatic fibrous dysplasia;[9] localized osteitis fibrosa cystica; subperiosteal hemorrhage of scurvy; and the classical sclerosing osteomyelitis of Garre.

We believe that it might be advisable to register all atypical inflammatory lesions of bone with the Bone Tumor Registry so that the peculiar characteristics of the nonspecific and specific lesions might be correlated just as the neoplasms of bone have been. This would also be of value in proper classification of the inflammatory group of bone lesions as well as that poorly understood group of bone lesions that are now being classified as "fibrous dysplasias."

SUMMARY

Three cases of somewhat different types of chronic inflammatory lesions are presented to show the difficulty frequently encountered in making a differential diagnosis between an inflammatory lesion and a neoplasm.

One of these cases looked much like a Ewing's tumor of the shaft of the tibia; the second case looked like an osteogenic sarcoma of the shaft of the metatarsal bone; and the third case resembled an osteogenic sarcoma of the end of the fibula in a child.

The differential diagnostic points in these cases are brought out by the case histories and discussions.

The importance of making a careful biopsy before instituting radical treatment in a case of suspected malignancy of the bone is pointed out.

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ABSTRACT OF DISCUSSION

Dr. J. L. Goforth, Dallas: These 3 cases not only illustrate well the difficulties which sometimes retard diagnosis in border line lesions, but also direct attention to the bizarre tissue reactions which may follow chemotherapy and antibiotic therapy. Certainly, as has been emphasized in the paper, the patient's interests demand always the first consideration; and when surgeon, clinician, roentgenologist, and pathologist work cooperatively together, the patient is given the best chance for the earliest institution of curative treatment. Diagnostic problems of this kind do not belong to any one specialty in medicine.

To the physician who assumes the responsibility of treating obscure bone lesions, the guiding information which may come from the detailed re-taking of history; from the more frequent use of laboratory aids in diagnosis, such as sedimentation rate determinations, special biochemical studies, and critical study of the differential blood count; and from cytologic and bacteriologic studies (which may be conveniently carried out at the time of taking a specimen for biopsy, will occasionally prove to be thoroughly satisfying. I know of one instance in which the diagnosis of eosinophilic granuloma of bone was established by cytologic study of material obtained by aspirating a rib lesion for culture purposes, and of another instance in which small fragments of granulation-like tissue, obtained from an iliac lesion at the time culture studies were being made, revealed osteolytic osteogenic sarcoma histologically. In this last case, the roentgenograms presented the characteristic picture of an inflammatory lesion.

LOW MORTALITY AMONG SPECIALISTS

The mortality rate of physicians who specialize is 30 per cent lower than that of general practitioners, according to a report by Louis I. Dublin, Ph.D., and Mortimer Spiegelman which appears in the August 21 issue of *The Journal of the American Medical Association.* The two statisticians are from the Metropolitan Life Insurance Company, New York City. Their report is based upon data from the American Medical Directory.

Commenting upon the lower mortality of specialists, they say that the rate "may arise from the self selection of the healthier physicians to continue their studies, from higher income levels, from more favorable conditions of work, and from better opportunities for better medical care.

PHYSICIANS NUMBER 199,755

The Bureau of Medical Economic Research of the American Medical Association estimates that there were 199,755 physicians in the United States as of June 1, 1948. Frank G. Dickinson, Ph. D., director of the Bureau, says in an article which appears in the August 21 issue of *The Journal of the American Medical Association* that this number is 17 per cent greater than the 170,163 physicians shown in the 1940 A.M.A. Directory. The population of the United States has increased only 12 per cent since 1940.

STUDIES IN THE LABORATORY DIAGNOSIS OF CHANCROID DISEASE

PRELIMINARY REPORT

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Studies on the laboratory diagnosis of chancroid were initiated this year at the Fourth Army Area Medical Laboratory, Brooke Army Medical Center, Fort Sam Houston, Texas, because our observation had been that the diagnosis was ordinarily determined by a process of exclusion, which consisted of repeated darkfield examinations for spirochetes. If results were negative, diagnosis of chancroid or lymphogranuloma venereum was made based on clinical manifestations.

Others have noted similar difficulties. For example, chancroid disease was a considerable problem in our occupational troops in the Philippines during 1945-1946. A study of contacts reported by Trussell[7] of the Nineteenth Medical General Laboratory in Manila indicated that many of the women demonstrated chancroid lesions which were negative for spirochetes on darkfield examination; findings on direct smear were typical of Hemophilus ducreyi. A Venereal Disease Commission,[10] recommended by Dr. Earl Moore, War Department consultant, confirmed Trussell's findings: the suspected lesions in the women were caused by H. ducreyi. Skin tests in both men and women with the Ducrey vaccine were usually positive in these cases where there was involvement of the femoral lymph nodes, but not where there were only early lesions of the genitalia. Other writers[4] point out that a lesion must be present at least five to fifteen days before the skin test becomes positive. The Commission found further that the lesions responded readily to sulfadiazine treatment, given locally or systemically.

Dr. O. Teague, an early worker with this disease in Panama, was one of the first to culture the organism from chancroid lesions, and used a medium composed of inactivated, clotted rabbit's blood.[8] The contaminating organisms with this medium were so numerous that the identification of H. ducreyi was made difficult. Various other culture media have been used, such as Martin's broth

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with 20 per cent defibrinated rabbit's blood added, and a modification of Besredka's egg medium. The tryptose phosphate agar blood medium developed by Difco has been found in recent years to be especially adaptable for the growth of fastidious organisms. Beeson and Heyman[3] reported this medium suitable for Ducrey, but noted that Ducrey's bacillus, unlike other Hemophilus organisms, will not grow with either or both X and V growth factors. Ducrey's bacillus needs whole blood or serum.[2]

After a trial of other media, the tryptose phosphate medium with fresh rabbit's blood was finally adopted for the study of patients with penile lesions referred to our laboratory for diagnosis. The objective has been to formulate new methods or improve known methods which would be practicable in the routine laboratory diagnosis of chancroid. It was anticipated that if a routine method of culture could be developed, comparison of the culture with the direct smear would aid in the bacteriologic diagnosis of the disease.

After repeated practice in comparison of smears from cultures and genital lesions, it has usually been found possible to make a diagnosis of H. ducreyi from the morphologic appearance of the organisms in smears. It was found early that the care taken in the preparation of the lesion for a smear and culture is the most important prerequisite for success. This is more important in lesions of longer duration than those in the early stages.

COLLECTION OF SPECIMENS

The technique used in preparing these lesions is to clean the ulcer carefully with gauze dipped in normal saline solution. The small superficial ulcer is entirely cleansed, removing the exudate. On the older and deeper lesions it has been found necessary only to clean and abrade the margins until they are free from necrotic tissue and exudate, causing them to bleed slightly. Approximately four to five minutes is required to prepare a lesion properly. Following the cleansing process, sufficient time is allowed so that the serum will form on the cleansed surface. At this time the material for the culture is taken, the procedure being to dip a sterile, platinum loop into the serum and obtain a small amount. This is then streaked directly to a freshly prepared rabbit's blood tryptose phosphate agar plate. The plate is streaked by a continuous horizontal movement from the top to the bottom of the plate without crossing or breaking. Growth usually occurs in the first half of the inoculated plate. A second plate of chocolate rabbit's blood agar is inoculated in a similar manner, and after this the following described liquid medium is inoculated.

Two to five impression smears are made on scrupulously clean slides; the lesion, which is held by the forefinger and thumb with a rubber gloved hand, is contacted with the slide. If an impression smear cannot be obtained, the serum is collected with a loop and dotted on the slide, avoiding streaking. Another technique, mentioned by Allison,[1] is to collect material from the overhanging edge of the margin of the ulcer with the flat end of a sterile toothpick, and to dot it on the slide, avoiding streaking. The smears are allowed to dry.

TABLE 1.—*Diagnosis on 40 Suspected Chancroid Cases.*

Case. No.	Darkfield	Serology	Lymphogranuloma Venereum C. F. T.	Ducrey Smear	Ducrey Culture	Diagnosis
1	—	—	—	—	—	Ulcer, penile
2	—	—	—	+	NM	Chancroid
3	—	—	—	+	NM	Chancroid
4	NM	—	NM	—	NM	NM (patient transferred)
5	—	—	—	+	+	Chancroid
6	—	—	—	+	+	Chancroid*
7	—	—	—	+	+	Chancroid*
8	—	—	—	+	+	Chancroid*
9	—	—	—	—	—	Ulcer, penile
10	—	—	—	+	?	Chancroid*
11	—	—	—	+	+	Chancroid*
12	—	—	—	+	+	Chancroid*
13	—	—	—	—	NM	Condyloma accuminata
14	—	—	—	+	+	Chancroid
15	—	NR	+	?	?	Lymphogranuloma venereum
16	—	—	—	+	—	Chancroid
17	—	—	—	—	—	Ulcer, penile
18	—	Kahn— Kolmer±	+	?	—	Lymphogranuloma venereum
19	—	—	—	+	+	Chancroid
20	—	—	—	—	—	Ulcer, penile
21	+	—	—	+	+	Chancroid and syphilis, primary
22	—	—	—	—	—	Ulcer, penile
23	—	—	—	+	?	Chancroid
24	—	—	—	—	—	Chancroid*
25	—	—	—	?	+	Chancroid*
26	+	NR	—	—	—	Syphilis, primary
27	—	—	—	+	NM	Chancroid
28	—	—	—	+	+	Chancroid
29	+	—	—	—	—	Syphilis, primary
30	—	—	—	+	+	Chancroid
31	+	—	—	—	—	Syphilis, primary
32	NR	+	—	—	—	Syphilis, primary
33	—	—	—	+	+	Chancroid
34	—	—	—	—	—	Ulcer, penile
35	—	+	—	—	—	Syphilis, primary
36	—	—	—	—	—	Ulcer, penile
37	+	+	—	+	—	Syphilis, primary, and chancroid
38	—	+	—	—	—	Syphilis, primary
39	—	+	—	+	+	Syphilis, primary, and chancroid
40	—	—	+	+	NM	Lymphogranuloma and chancroid

NM—Not made.
NR—Not reported
*Ducrey skin test positive. Reports of skin tests not available in the majority of cases.

Chancroid	19
Syphilis	6
Lymphogranuloma venereum	2
Chancroid and syphilis	3
Chancroid and lymphogranuloma	1
Undiagnosed	8
Condyloma accuminata	1
Total cases	40

Serum is collected weekly, when possible, from the patient for future laboratory work such as agglutination and complement-fixation tests, which have been attempted by some workers in the past but have not proved successful.

If the original culture is negative, an attempt is made to collect at least two or more follow-up cultures.

SMEARS

Burke's modification[6] of Gram's stain is used in preparing the direct smears for microscopic examination. Ducrey's bacilli do not absorb the counter stain readily, and this characteristic aids in the differentiation from other gram-negative organisms. This fact should be emphasized. On smears, the organisms are found singly, in pairs, and in short chains. The organisms are gram-negative, pleomorphic, sometimes found as cocci, diplococci, or small diplo-bacilli, and sometimes appear boat-shaped. Occasionally they are found within the white cells, and infrequently within or on the epithelial cells. The individual organisms in chains usually follow the mucous shreds in the smears. We have found that the typical "railroad tracks" and "school-of-fish" formations, contrary to common belief, are exceptions and are rarely seen. However, when such a formation is present this is a good criterion for diagnosis.

MEDIA

The following described media are those used routinely in the chancroid studies in the Fourth Army Area Medical Laboratory:

1. *Tryptose Phosphate Agar Base for Blood Plates*[5].—Tryptose 20 Gm., sodium chloride 5 Gm., sodium phosphate 2.5 Gm., dextrose 1 Gm., distilled water 1,000 cc., and agar* 18 Gm. at a pH of 7.4 to 7.6 are heated to dissolve. The material is autoclaved at 15 pounds pressure for fifteen minutes. It may be stored in small amounts (250 cc.) in Erlenmeyer flasks.

To make blood plates from stored flasks, the flasks are placed in a water bath and the media boiled until it is completely dissolved. The media is then cooled to from 45 to 48 C. and enough blood added to make 5 per cent of the total volume. Rabbit's blood is superior, but horse's blood may be used.

2. *Tryptose Phosphate Agar Base for Chocolate Agar Plates.*—The procedure is the same as above except for the addition of 15 Gm. of proteose-peptone no. 3 per 1,000 cc. of the media above. The mixture is boiled in a water bath as above and cooled to 60 C. Enough blood is added to make 5 per cent of the total volume. The media is then placed in a water bath and heated slowly to from 80 to 85 C., rotating to produce an even mixture. The media is cooled to 55 C. and slants or plates are made. They are incubated overnight and checked for sterility. They should be stored in a refrigerator.

3. *Liquid Medium.*—Sterile sodium chloride, 85 per cent, 200 cc.; sterile beef infusion broth 25 cc.; sterile rabbit's serum 25 cc.; and sterile rabbit's blood, "laked," 10 cc. are checked separately for sterility before use. This may be done by dividing a blood agar plate into several sections and streaking each part to a section, incubating the blood plate, and checking for sterility.

"Laked" blood is prepared as follows: Three cc. of sterile defibrinated rabbit's blood is added to 7 cc. of sterile distilled water. It is allowed to "lake" for fifteen minutes, is centrifuged, and the supernatant fluid added to the medium.

The medium is tubed in 5 cc. amounts, incubated for sterility, and stored in a refrigerator.

*Difco tryptose phosphate broth is used. Agar is added by this laboratory.

CULTURES

The cultures are incubated in candle jars containing approximately 10 per cent carbon dioxide and a high moisture content (obtained by the use of saturated cotton or sponges). This laboratory has had the best success at incubation at from 36 to 37 C., although some authors[3, 9] use the temperature of 34 C. The cultures are examined daily, and organisms from the broth medium are subcultured to the chocolate agar plates. The reason for subculturing immediately is that contaminants present may over-grow the colonies of Ducrey. On solid media, the colonies appear small, convex, clear, and almost colorless; they are round and have entire edges, taking on a dewdrop appearance. On blood agar the colonies slip away from the loop and slide over the surface, and do not assume the satellite phenomenon typical of other Hemophilus organisms. After from three to five days the colonies become larger, showing a slight pigmentation and taking on a crateriform depression; they also show a slight hemolysis. On chocolate agar plates the hemolysis assumes a slightly green discoloration. The morphology of the smear taken from organisms grown on solid media is as follows: At twenty-four hours the organisms tend to be in short chains which usually lie in parallel formation; after forty-eight hours the organisms become more granular and are found singly and in pairs rather than in chains. The organisms are not in parallel formation but in clumps. Burke's modification of Gram's stain is used except that carbol fuchsin is used as a counter stain, rather than safranin. Carbol fuchsin is more penetrating and allows a closer detail study of morphology. In liquid media the organisms tend to form long chains, referred to as tangled masses.

MUTATION

The liquid medium described was devised in an attempt to obtain a single medium useful for many purposes: (1) for original isolation, (2) for maintaining stock cultures, and (3) for in vitro antibiotic sensitivity determinations.

In preliminary trials with this medium, it was noted that growth after subculture from chocolate agar was quite satisfactory. However, a peculiar and apparently hitherto unreported phenomenon was soon observed. If growth is allowed to continue for from twenty-four to forty-eight hours and subculture is then made to chocolate agar, the colonies on the latter medium have the typical appearance of H. ducreyi. On the other hand, if growth in the liquid medium is allowed to continue for from four to five days, a subculture to chocolate agar at this time reveals an apparent mutation. The colonies are large, yellow, mucoid, and have a glistening sheen. Except for the glistening sheen and a slightly darker pigmentation, they look much like Staphylococcus aureus. For this reason the colonies were originally thought to be contaminants, but it soon became apparent that they were probably mutations. Up to the present time three separate batches of media have been used; in all cases where subculture from broth to chocolate agar has been made, the mutation has appeared, both in cultures isolated from patients and in "known" stock strains. Smears from the "mutant" colonies show organisms mor-

phologically identical (except for slightly larger size) with Ducrey's organisms from typical colonies. Obviously, too little work has been done to claim this as an established mutation, but it is an interesting phenomenon. Studies are being continued.

SUMMARY

1. In this laboratory tissue impressions on glass slides properly made and stained from chancroid ulcers have consistently demonstrated morphologically characteristic H. ducreyi organisms.

2. Of 40 penile ulcers suspected to be chancroid, 21 were found positive by direct smear; 34 of the 40 cases were cultured, and 14 of these were positive for chancroid.

3. It is our experience that the impression smear and culture are reliable. It is stressed that the method of preparation of the lesion for making the smear is essential for success in making a good impression slide, and for obtaining cultures of the organism.

4. Confirmation of the value of the tryptose phosphate agar medium has been shown.

5. An apparent mutation form of H. ducreyi, hitherto undescribed as far as can be determined, has been reported.

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ORAL HYGIENE SUFFERS

Four out of five of the toothbrushes now in use in American families are so badly worn or in such an unsanitary condition that they are no longer useful for oral hygiene, according to a survey reported upon in the September issue of the *Journal of the American Dental Association.*

The study, conducted through a group of families representing a cross section of the American population, revealed that 80.7 per cent of the toothbrushes in use by family members were in need of replacement because they could no longer be effective implements in mouth hygiene or because their use might injure dental tissues. Approximately three-fourths of the unsatisfactory brushes were found to have bent, broken, or matted bristles. The remaining one-quarter of the unsatisfactory toothbrushes, or one out of each five examined, were found to be unsanitary.

TULAREMIA

SOME CLINICAL AND PATHOLOGIC OBSERVATIONS

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Tularemia was first classified by the United States Public Health Service in 1927 as one of the reportable diseases. Its identity and etiology were not well established until 1920. Prior to 1924, only 15 cases with 2 deaths had been so diagnosed and reported in the United States. During 1924, 1925, and 1926, 308 cases with 11 deaths were reported. In 1927, 8 cases of tularemia were reported from Texas. Figure 1 shows the number of cases reported yearly, from 1927 to 1946, in Texas and the adjoining states of Oklahoma, Arkansas, and Louisiana.[8] No doubt there are cases of tularemia occurring now within Texas that are not reported to the State Health Department. The increase of frequency and the high mortality of tularemia in the Southwest have served as a stimulus for us to study this disease.

The first report of what we recognize now as tularemia was made by Pearse from Utah in 1910. He reported 9 cases as "deer fly fever." Three years before, Martin of Arizona wrote to Novy in Michigan and described 5 cases in which he considered the disease to result from skinning and dressing wild rabbits. McCoy and Chapin, in 1911, isolated Bacterium tularense from ground squirrels in Tulare County, California. Vail of Cincinnati in 1914 reported the first human case of tularemia in which B. tularense was isolated.[6]

Tularemia usually manifests itself in one of the following four ways: (1) ulceroglandular, characterized by an ulcer of the skin usually located upon the fingers or hands, accompanied by enlargement of the epitrochlear and axillary lymph nodes; (2) oculoglandular, in which a lesion occurs on the eyelids, accompanied by a conjunctivitis and enlargement of the regional lymph nodes; (3) pneumonic, with signs of toxemia and sepsis with either lobular or lobar involvement of the lungs; (4) typhoid, which clinically resembles typhoid fever. The latter variety may either follow any one of the other three forms of the disease or it may be a specific form of tularemia. In such cases lesions are distributed throughout the body.

DIAGNOSIS

A positive diagnosis of tularemia, of course, may be made either from a culture or by serum agglutination. The culturing of

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these bacteria is not practical since the handling of the organisms in a laboratory is unwise because many cases of tularemia have developed in laboratory workers.[6] The agglutination titer may be too low to be significant before the twelfth day. An increase in the titer, of course, is significant. It may reach 1:1,250 in the third week of the disease. Skin reactions to specific sera may be of diagnostic value. Cross-agglutination occurs with Brucella melitensis so that this disease may be confused with tularemia.

The history of contact with wild rabbits is important in arriving at a diagnosis in the ulcerative forms of tularemia. The disease is frequently transmitted to man by the deer fly,[3] the wood tick, and the rabbit tick. The ground squirrel, water rat, jack rabbit, dog, and possibly the sheep muskrat, opossum, woodchuck, rabbit, and grouse may serve as animal reservoirs, and possible sources of infection for man, according to Moore.[5] The common routes of infection for man are through the skin, conjunctiva, and sometimes the gastro-intestinal tract. The incubation period is about three days. The disease usually runs its course in from two to three weeks; however, some patients are sick from two to three months. The disease is considered to produce a lifetime immunity.

The infectivity of B. tularense for laboratory workers cannot be over emphasized. Two pathologists are known to have developed the disease from the handling of infected material at autopsy. One of these was an associate of mine. Although he was not performing the autopsy, in his enthusiasm to see and demonstrate the lesions to the students, the infected material was handled without gloves. Approximately seventy-two hours later, a small infected hair follicle was observed on the middle finger of his left hand. During the subsequent forty-eight hours, there was general malaise and a gradual enlargement of the lymph node in the cubital fossa and axilla on the left. Although these lymph nodes became large, tender, and fluctuant, suppuration did not occur. The primary lesion healed without significant ulceration. The lymphadenitis, fever, and general malaise persisted for several weeks. Streptomycin was given. The agglutination titer was 1:1,250 during the third week of the disease. It was approximately four months, however, before the patient felt that he had completely recovered from this infection.

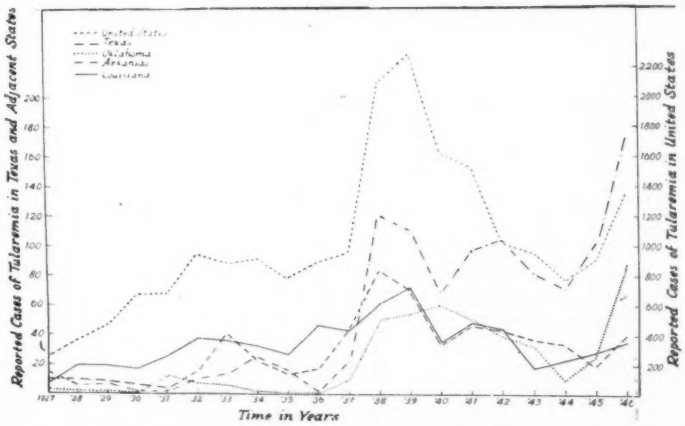

FIG. 1. Cases of tularemia reported between 1927 and 1946 to the U. S. Public Health Service from Texas, Oklahoma, Arkansas, and Louisiana.

TREATMENT

Many different forms of therapy have been used in tularemia. The intravenous use of bismuth sodium tartrate has been advocated.[4] Bell and Kahn[1] treated laboratory animals with tularemia with sulfanilamide, sulfadiazine, sulfamerazine, acriflavine, metaphen, iodide and bismuth, trivalent arsenic alone, arsenic and bismuth, antimony, penicillin, and hyperimmune equine antitularemic serum. These investigators concluded from their experiments that none of these drugs had any appreciable effect upon the disease with the possible exception of penicillin.

Foshay, a long advocate of immune sera in the treatment of tularemia, recently reported that immune serum, if used early and in a sufficient dosage, is of definite benefit and hyperimmune serum is more beneficial. However, in his more recent use of streptomycin, it was found to be better than sera. Foshay said that "one-half million units of streptomycin daily for two days, followed by one-fourth million units daily for four days might well become the provisional basic dosage for human tularemia."[2] It appears at this time that streptomycin is the agent of choice for the treatment of human cases of tularemia.

SUMMARY OF CASES

At the University Hospital in Little Rock, Ark., 26 cases of tularemia were seen between January. 1940, and July, 1946.* In

*These cases were reviewed during the time I was associated with this hospital.

this group there were 18 cases of the ulceroglandular type, 3 of the pneumonic form, 2 each of the oculoglandular and typhoid types, and 1 case was unclassified. The disease may occur at any age. In this group, 4 cases occurred before 9 years of age, 6 between 10 and 19, 2 between 20 and 29, 7 between 30 and 39, and 3 between 60 and 69 years of age. White males had the disease more often than either females or Negroes. A total of 21 cases occurred in the white race and 5 in the Negro; 22 of these were in males and 4 in females. One-third of the group gave a history of some contact with rabbits; 3 patients said they were bitten by a tick; others attributed the occurrence of tularemia to the bite of either a cat or a skunk; while others did not associate their illness with any vector. One of our cases resulted from an infection occurring at an autopsy on a case of tularemia. Four of the 26 patients with tularemia died from the disease and each was subjected to autopsy.

Many of the patients were unable to give the exact time of onset of their infection. However, a few placed it as from one to twenty-one days before they were admitted to the hospital. Only 2 patients were sick for less than twenty days; 18 were sick from twenty to fifty days; while 1 was sick for three months. The diagnosis of tularemia in 21 of the 26 cases was confirmed by a positive agglutination test, and in 4 by an autopsy. One case was diagnosed only from the clinical findings.

The following cases from the University Hospital series illustrate the clinical findings in each of the four types of tularemia.

CASE REPORTS

Ulceroglandular Type.—The patient was a white woman 35 years of age. She dressed two rabbits twenty-five days preceding the time of hospitalization. At that time she punctured the skin of the right index finger in two places. Three days after dressing the rabbits she developed a headache, nausea, rigors, and swelling of the right epitrochlear and axillary lymph nodes. Approximately one week following the injury to her right finger, the areas were swollen and red. Shortly thereafter they became necrotic and subsequently developed into two punched out ulcers. These lesions healed before the patient was hospitalized. Chills, fever, nausea, vomiting, pain in lower extremities, headaches, and loss of weight had been experienced by the patient. The fever subsided, however, four days before she came to the hospital. Two days before admission, she developed a skin eruption over the extremities.

On admission she was not acutely ill; however, the rash was present in the skin of the forearms and on the anterior surface of the legs. It was macular, slightly elevated, nonpainful, and did not itch. The skin lesions disappeared when the sulfa drugs were discontinued. The healed ulcers on the index finger were approximately 3 mm. in diameter. The epitrochlear and axillary lymph nodes were enlarged two to three times their normal size. The white blood cell count was 10,200 with 39 per cent polymorphonuclear leukocytes, 52 per cent lymphocytes, 6 per cent stabs, and 3 per cent eosinophiles. The hemoglobin and red cell counts were normal. The urinalysis was negative. The agglutination test for tularemia was positive in a dilution of 1:640.

The patient remained in the hospital for seventeen days. The temperature was elevated during the first five days after which it remained approximately normal. The axillary lymph nodes remained enlarged and tender but did not appear as if suppuration was going to occur. The lymph nodes were still enlarged when she was discharged from the hospital.

Oculoglandular Type.—The patient was a white boy 13 years of age who was admitted with an infection in the left eye and a history of fever for two weeks. He thought that a tick bit him on the upper left eyelid about thirty days previously. Two weeks later there developed an acute purulent conjunctivitis in the left eye. This was followed by cervical lymphadenitis. A chill, fever, and vomiting accompanied this ocular infection. The fever persisted. The infection in the left eye improved following the use of "eyewash." However, the cervical adenitis persisted.

On admission the conjunctiva of the left eye was hyperemic and the upper lid was edematous. The cervical lymph nodes on both sides were enlarged and tender. This was more marked on the left side. The lymph nodes in the left axilla were enlarged. The spleen was palpable. There was an acute pharyngitis and acute otitis media on the left. A fine macular rash present in the skin over the lower thorax, abdomen, and lower extremities faded on pressure. The temperature was 104 F., pulse 125, and the respirations were 28 per minute. The red blood cell count was 3,650,000, the hemoglobin 76 per cent, and the white blood cell count 12,000 with 69 per cent polymorphonuclear leukocytes, 20 per cent lymphocytes, 6 per cent stabs, 4 per cent monocytes, and 1 per cent eosinophiles. Urinalysis was negative.

Numerous leukocytes were present in a smear from the left eye; however no bacteria were demonstrated. A blood culture showed no growth after twenty-four hours incubation. A smear from the throat showed bacteria consistent with nonhemolytic streptococci. On the fourth day after hospitalization agglutinations for protein OX 19 were negative, while the agglutination for B. tularense was positive in a dilution of 1:160. The titer for B. tularense was 1:640 ten days later.

The patient remained in the hospital for twenty days during which time the temperature gradually returned to normal. The cervical adenitis decreased.

Pneumonic Type.—The patient was a Negro man 65 years of age who was unable to give any history when brought to the hospital by his daughter. She stated that he was in good health until five weeks before, at which time he had a chill and a headache. The patient remained in bed because of weakness, headache, cough, and fever. The cough increased in severity and it became productive of a thick, yellowish-white sputum. He became worse and, during the two weeks before admission, was unable to talk. The fever continued with what was considered to be afternoon elevations. The patient lost 50 pounds of weight during the five weeks of illness.

On examination the patient was emaciated and dehydrated. The respirations were rapid and shallow, and the skin was warm and dry. Bronchial breath sounds were present throughout both lungs. Coarse, moist rales were present in the left subclavicular area; no friction rub was observed. The heart rate was 120 per minute, the sounds were distant, no murmurs were heard. The nonprotein nitrogen was 70 mg. per 100 cc. The blood serology was positive. The red blood cell count was 3,770,000, and the white blood cell count was 7,100 with 68 per cent

polymorphonuclear leukocytes, 19 per cent lymphocytes, 6 per cent stabs, 4 per cent juveniles, and 3 per cent mononuclears. The temperature was 100 F., following which it dropped to 99 F. at the time of death.

PATHOLOGIC EXAMINATION.—The autopsy was performed five hours following death. The anatomic diagnosis was as follows: Tularemic pneumonia involving both lungs; focal necrosis in liver and lymph nodes; pleural effusion, bilateral 100 cc.; ascites 100 cc.; bronchiectasis, acute and chronic, bilateral; generalized atherosclerosis, old cerebral hemorrhage in the area of the left internal capsule; emaciation; dehydration.

Although the pulmonary lesion in this case was consistent with the diagnosis of tularemia the diagnosis might be questioned in the absence of a significantly positive agglutination reaction and the failure to demonstrate the organisms in culture. However, in view of the fact that one of the pathologists developed an ulcer on the finger followed by axillary lymphadenitis with an agglutination reaction of 1:1,280 to B. tularense, the original diagnosis was confirmed.

Typhoid Type.—The patient was a white man 35 years of age. He had worked as a farmer and his health was good. Ten days before admission to the hospital he went to work in the morning and about 10 o'clock he felt tired and ached all over. He stopped work. During the afternoon he developed a headache and anorexia. At this time, nocturia occurred. Urination was accompanied by frequency and burning. During the following four or five days he had fever and night sweats, but no chills. All of his bones seemed to ache. There was pain in the lower lumbar region. Essentially nothing could be retained by mouth.

A nonproductive cough developed two or three days before hospitalization. This was accompanied by a sensation of tightness in his chest. There was some jaundice at this time. There was nothing in the history to suggest tularemia.

On admission to the hospital the patient was acutely ill, with a temperature of 101.4 F., pulse 84, and respirations 16 per minute. The white blood cell count was 6,700 with 63 per cent polymorphonuclear leukocytes, 24 per cent lymphocytes, 6 per cent mononuclears, and 4 per cent stabs. The red cell count and hemoglobin were normal. The urine was normal. Two lymph nodes were enlarged in the right inguinal region.

A friction rub was present at the base of the right lung seventy-two hours following admission, and forty-eight hours later there was clinical evidence of pneumonia in both lower lobes. The cough was still present; however it was nonproductive. The patient continued to run a high spiking temperature reaching 104 and 105 F. Two days before death, he had his first chill and, the following day, a purpuric, minute rash appeared in the skin over his lower chest and abdomen. This rash did not disappear on pressure.

LABORATORY EXAMINATION.—Several blood cultures were negative. Agglutination for B.typhosum was positive in 1:40 dilution, for undulant fever it was negative, and for B.tularense, positive in a dilution of 1:320 on the day preceding death. The leukocyte count was slightly elevated during the first three days. However, it dropped from 12,800 on the third day to 7,900 on the fourth day, and to 5,700 on the seventh day of hospitalization. The polymorphonuclear leukocytes varied from 63 to 84 per cent. The blood serology was negative. A few red and white blood cells were present in the urine, the albumin was 2 plus, and the specific gravity varied between 1.012 and 1.025.

PATHOLOGIC EXAMINATION.—The autopsy was performed one hour after death. The changes were typical of a widespread tularemic infection. The anatomic diagnosis was as follows: Tularemic pneumonia involving both lungs; empyema, bilateral (500 cc.); mucus plugs in bronchi; atelectasis of lungs; acute generalized peritonitis (1,000 cc.—no bacteria demonstrated); splenomegaly 210 Gm.; focal areas of necrosis in spleen and liver (tularemic); focal areas of necrosis in inguinal, mesenteric, retroperitoneal, and mediastinal lymph nodes (tularemic); cyanosis of lips; emaciation; ulcerations in mucosa of stomach and rectum secondary to stomach and rectal tubes; hemangioma of liver.

DISCUSSION

The frequency of tularemia in the Southwest, as reported to the State Health Department, and the marked increase in the mortality rate, as observed in a series of 26 cases seen at the University Hospital in Little Rock, Ark., indicates the growing importance of this disease. The mortality rate varies from 3.7 to 11 per cent;[6, 7] however, in this group of cases it was 15 per cent.

There is a wide variation in the clinical manifestations of tularemia. The typhoid and pneumonic types of the disease may be confused clinically with several of the febrile diseases. The diagnosis usually may be made on the basis of a specific agglutination reaction; however, in fulminating cases the disease may not be recognized until an autopsy is performed.

No doubt there are clinical cases of tularemia in which the disease is so mild that it passes unnoticed. Some of the patients included in this group show that tularemia may follow a protracted course for as long as three months. Such a prolonged disease as this becomes of increasing economic concern.

B. tularense is usually transmitted to man either through the handling of infected animals, such as squirrels and rabbits, or through the bite of certain blood sucking vectors. The wood tick is considered to be the natural host and the organism is passed to successive generations by way of its eggs. The preservation of the wood tick by the prevention of forest fires may be a significant factor in the increased incidence of this disease in some sections of the Southwest.

B. tularense usually enters the body through the skin and conjunctiva. Cases apparently may develop following the ingestion of infected meat insufficiently cooked. The primary lesion on the skin usually begins as a papule which is followed by ulceration. A local lesion in the skin may develop within from forty-eight to seventy two hours following infection. It usually progresses to an ulcer with a firm and elevated margin and a necrotic base. In a short time the regional lymphatics become swollen, painful, and sometimes suppurate. Agglutinins usually appear in the blood during the second week

of the disease and persist for an indefinite period.

Most of the cases of tularemia in this series were treated with sulfa drugs. The fact that a majority of these patients recovered is not indicative of the value of these drugs in the treatment of this disease. Bell and Kahn[1] found that the sulfa drugs did not have any effect upon tularemia in experimental animals. No doubt there are many people who have this disease and recover without receiving any therapy. The more recent studies would indicate that streptomycin is the drug of choice in the treatment of tularemia.

SUMMARY

Tularemia is discussed from a clinical standpoint with regard to diagnosis and treatment, and from the frequency of the disease in the Southwest. A group of 26 cases is reviewed. These data would indicate that tularemia is becoming more frequent, and the mortality rate is higher in this series than that previously reported by others. A case is reported to illustrate each of the four types of the infection as observed in man.

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ABSTRACT OF DISCUSSION

Dr. C. T. Ashworth, Dallas: It may be worth while to consider briefly the nature of the reaction of the body to the B. tularense. The inflammatory reaction which occurs in this disease has been noted to resemble that seen in the group of infectious granulomas, large mononuclear wandering cells and epithelioid cells, members of the reticulo-endothelial system, being prominent in the cellular response. The focal nature of the reaction and the occurrence of necrosis in the center of the lesion, normally followed by suppuration, are also characteristic of infectious granulomas. Because of these features the physician may have the problem of attempting to differentiate the lesions of tularemia from those of acute miliary tuberculosis, sporotrichosis, undulant fever, histoplasmosis, and others. The suppurative granulomatous lymphadenitis which occurs in lymphogranuloma inguinale may be similar to the lymph node lesions seen in tularemia. In all these cases, assiduous examination for specific causative organisms, serologic and skin tests, and particularly the clinical findings will be of particular importance when considered in conjunction with the morphologic characteristics of the lesions in establishing the diagnosis.

I am particularly glad that Dr. Rigdon has emphasized the danger of infection occurring in pathologists and others who come in contact with the diseased tissues of patients who die from this disease. I am afraid that some of us may fail to exert the proper precautions in performing autopsies in infectious cases of this sort; this I believe to be true particularly in the case of patients who die from tuberculosis. I would join Dr. Rigdon in urging that anyone who performs an autopsy upon a person suspected of having tularemia exert the utmost precautions in preventing dissemination of the infection. The same note of precaution should also be applied to those who attempt to culture the organisms, since, as is well known, a considerable number of laboratory workers have unwittingly infected themselves from bacteriologic preparations of the B. tularense. Many good clinical laboratories will not endanger the workers by attempting to culture the B. tularense, depending for diagnosis instead upon the agglutination test.

I am especially interested in the graph which shows the incidence of tularemia in Texas and neighboring states, and I want to ask Dr. Rigdon if from his studies on the epidemiology of tularemia he can explain if the peak in incidence shown to occur around 1939, and possibly the peak of 1945-1946, are of statistical significance and if so how they may be accounted for. Also, I would like to know what may be the explanation for the high incidence of the disease in Arkansas. The graph shows from 5 to 10 per cent of the cases in the United States since 1938 to have occurred in this state. Were this corrected for population, it would indicate an even higher incidence than shown.

Dr. Rigdon, closing: I am not enough of a statistician to interpret the significance of the data in the graph. I can only say that apparently from these figures we may have an increase in the frequency of tularemia within a short period of time. The number of cases in Texas and Louisiana is not increasing at a rate similar to that in Arkansas and Oklahoma. The total number for the United States apparently is on the increase. Why the frequency of this disease is greater in Arkansas than in Texas apparently may be explained on the basis of the wood tick. In view of the extensive forests in Arkansas and the effort to protect these from fires, we may be preventing the killing of large numbers of ticks which may be responsible for the high incidence of occurrence of this disease.

I cannot overemphasize the significance of the possibility of infections developing in laboratory workers. Apparently the organism may enter the unbroken skin. The case referred to in this paper of the pathologist who developed the infection from handling autopsy material developed the lesion on the finger, and as far as he knows, there was no abrasion in the skin at the time of infection.

To advocate the indiscriminate use of streptomycin, especially in moderately advanced or advanced cases of pulmonary tuberculosis, not only is premature but also carries with it certain dangers and drawbacks. Among the principal dangers in the use of this drug is its toxicity, which may seriously affect hearing, sight and kidney function and cause skin eruptions. At present, it can only be said that we have seen little in the treatment of well established pulmonary tuberculosis by streptomycin that gives cause for any great optimism regarding its curative value.—Comm. on Tuberc., New Hampshire Med. Soc., New England J. Med., Oct. 23, 1947.

TEXAS PUBLIC HEALTH TRAINING UNIT

B. M. PRIMER, M. D.
Director Travis-Bastrop County Health Unit
AUSTIN, TEXAS

At a conference in the fall of 1946 between representatives of the Texas State Department of Health, the Kellogg Foundation, and myself, the need was discussed for providing a short training period for new employees of the State Health Department and refresher courses for personnel now employed in health departments throughout the state. It was suggested that the Travis-Bastrop County Health Unit would be a desirable center for such a training course on account of its proximity to the central office of the State Health Department and because its size and organization are sufficient to be more easily adapted to this type of program.

Following this meeting, plans together with a budget were submitted to the Kellogg Foundation for approval. These were approved on a five year basis and the Training Unit was started as of January 1, 1947. During January, a stenographer was employed and preliminary work was started on securing additional office equipment and supplies. A sanitary engineer and an assistant director of nurses were employed and tentative schedules were drawn up for a six weeks training program for sanitarians and nurses. Since this was a new program to most of us, schedules were arranged so that they could be modified to suit the situations as they developed. Field training for nurses had been in operation since 1943 on a cooperative basis with Incarnate Word College of San Antonio, but it was necessary to adapt this program from a four months plan to one of six weeks and coordinate both plans with our regular public health nursing program. The program for sanitarians was developed from the expressed desires of the State Health Department and the needs of the students as noted by the instructors in the first few classes.

During the year all grades of sanitarians and nurses have attended the training school. Some have been people of long experience but no training, others of good training but little experience under supervision, and some with little or no experience and no training. Few of the physicians had previous experience or training. During the first year of operation there were enrolled in the school 6 physicians, 21 nurses on six weeks training period, and 5 on three to four months training, 40 sanitarians, and 4 clerks.

Read before the Section on Public Health, State Medical Association of Texas, Annual Session, Houston, April 27, 1948.

HEALTH OFFICERS

Physicians entering the service of the State Health Department received orientation training (approximately one week) in the office of the director of Local Health Services, during which time they visited all of the divisions and bureaus of the State Health Department and were instructed in the different activities and services. Following this period they spent several weeks in the Public Health Training Unit under the supervision of the director. This training was carried out on an individual basis after consultation with each physician and determination of his needs. Daily conferences were held with physician trainees. Discussions covered the entire field of administrative practice including analysis of local health problems, methods of solving them, public relations, liaison with official and nonofficial agencies, and so forth. Physicians' schedules were arranged so that they would have an opportunity to observe those activities of a health program which they needed either to see for the first time or on which they needed to refresh their memories. None of the physicians had had formal postgraduate training in the public health field, although some (in their past experiences) had acted as part-time consultants in various capacities with health units. For this reason, greater emphasis was placed on improving their knowledge of sanitation and those subjects not familiar to most medical personnel. All trainees visited in the training school for sanitarians and made field trips with them. Daily discussions with each of the trainees on the previous day's work helped to impress on him his relationship with other members of the health unit staff and his responsibilities for direction and leadership in the unit.

PUBLIC HEALTH NURSES

Nurse trainees have all received essentially the same type of basic indoctrination. Following their arrival at the Training Unit, they received instruction in the organization and policies of the local health unit. They were assigned to a nurse supervisor and were under the guidance of this supervisor throughout the training period. All were given opportunity to observe and participate in the following types of activities: field trips, clinics (venereal disease, tuberculosis, immunization, maternity), home visits, conferences (well child conferences), schools, antepartum classes, family case conferences, and weekly staff education programs.

In field trips they visited such places as the State Health Department, State Department of Public Welfare, local dairies and

pasteurization plants, water and sewage plants, State School for Blind, State School for Deaf, State Department of Vocational Rehabilitation, State Public Health Laboratory, and Rapid Treatment Center at San Antonio.

Nurse trainees received instruction under supervision in all types of home visits. On the first visit, one of the senior nurses of the Health Training Unit demonstrated the correct technique to be used in making the visit. This was followed by several visits by the trainee alone and then by a supervised visit. At this time, an analysis was made of the student's ability, giving consideration to such things as plan or organization of work, approach to families or home, nursing technique, teaching ability, record keeping, appearance and manner, and strong points.

Close supervision of all trainees is the keystone of the nurse training, and trainees were checked by supervisors frequently to see if they understood the various procedures recommended in different activities. For example, in the procedures involved in a well child conference the following points were covered by the supervisor: clinic set-up, patient admission, weighing and measuring, taking temperatures, interval history, immunizations, conferences with mother following physician's examination, demonstrations, and records.

All trainees prepared cases for the family case conference and most of them presented the cases to the nursing staff. This was done to show the student the value of a careful family case study and to develop ability in presentation before a group of such a study.

SANITARIANS

Six sanitation classes were completed during the first year. The instructor personnel responsible for training consisted of a sanitary engineer and a sanitarian who worked under the director of the Training Unit. Trainees enrolled included personnel interested in making public health a career and who desired employment in the sanitation field with a city, county, or state public health agency as well as those now employed in public health departments as sanitarians or sanitary engineers who wished to receive refresher training in their respective fields.

The type of training presented to each class was determined largely by the qualifications of each group accepted for training. Although it is the consensus of those participating in this Training Unit that the training should be the applicatory type, it was found necessary to provide a full six week schedule for certain classes which needed detailed instruction in the basic fundamentals of sanitary science. Other groups represented by students who had varied experiences in the sanitation field were given only a three week schedule of didactic training supplemented by a three weeks period of applicatory field work. The didactic part of the program covered a discussion of such subjects as organization of health departments on the national, state, and local levels, insects and their control, rodent control, methods of food and milk control, swimming pool sanitation, methods of excreta and garbage disposal, water supplies, industrial hygiene, and so forth. Applicatory activities included making sanitary surveys and solving specific sanitary problems such as elimination of minerals from drinking water supplies. Every effort was made to conduct the training on a practical basis in order that the trainee personnel might learn to cope with the public health problems that arise in the average community. It was believed that "learning by experience" is far more valuable than the didactic method of instruction and that this type of training should be emphasized. With this policy in mind, the training course was organized so that at least 50 per cent of the time spent in training was devoted to field work and field projects. Each class was encouraged to select some project such as a sanitary survey or a community wide sanitation program in order to give training in community organization procedure and to enable the trainee to acquire skills in community planning and supervision of voluntary agencies and individuals who might be used in assisting him in carrying on his part of the health program.

During each week of training, students were assigned articles from various periodicals as special reports which were presented to the class. It was found that this type of training was extremely instrumental in promoting class discussions and it afforded the trainees an opportunity to become familiar with current publications and literature pertinent to the particular field of sanitation involved. These reports were made a part of the permanent Training Unit records and served as a means of evaluating student accomplishments.

All of the usual tools now available in the educational process were used in the training. Many excellent films, film strips, and literature were available either from official health department sources or were obtainable from commercial firms. Display material furnished by commercial sources on such subjects as insecticides, small unit chlorination systems, and other apparatus of a sanitary

nature proved useful in furnishing demonstrations for the trainees.

CLERKS

Originally it was planned to conduct a course for clerks which would cover a four weeks period. However, because of difficulties encountered in providing substitutes for clerks in smaller units and in financing them during their training period, it was necessary to shorten this period to one week. Only one class was held during the year and it was supervised by personnel furnished by the State Health Department through the Division of Local Health Services. Four clerks selected from four different health units spent a profitable week in the Training Unit. Subjects covered in the training program included such things as the organization of health departments on the national, state, and local level, record keeping in local health units, communicable disease files, tuberculosis files, the central tabulating unit of the venereal disease division, nursing records and files, sanitation records and files, the evaluation schedule, administrative records and reports, vital statistics records and their uses, qualification of a good clerk, and so forth.

The Manual of Records and Procedures for local health units was used as the chief source of information. Complete files for the average health unit were set up by the trainees during the training period and each of them was given actual practice in carrying on the various duties of a clerk. Discussions which took place on the various topics covered in the training period indicated the intense interest of clerks in acquiring more knowledge of the health program and the need of all health unit directors to devote more time to this important person in the health unit set-up.

FUTURE TRAINING

Because of lack of medical assistance in the Training Unit during the first year, it was found impracticable to give medical officers much applicatory training. Plans in the future call for placing them during part of their training in the Bastrop office of the Health Unit and supervising their operation of a health unit under rural conditions. Emphasis on sanitation and supervision of sanitarians will be continued.

In considering plans for future training, efforts have been made particularly in the program for nurses and sanitarians to determine what the student trainees thought they needed most and in what manner this material or instruction might be best given to them. Each nurse and sanitarian trainee was asked at the end of the training period to give his opinion regarding the training and he was encouraged to be frank in his remarks.

From a review of this information it was found that sanitarians should be given additional periods of applicatory training and that actual equipment such as dishwashing machines, chlorinators, and so forth should be available for instruction. It is believed that such equipment can be secured from commercial organizations and installed for experimental and teaching purposes. Plans have been made to provide an insectarium so that trainees may have an opportunity to observe the stages in the life cycle of insects of a public health significance and produce enough insects to serve as a means of demonstrating the effectiveness of various types of insecticides.

Nurse trainees believed that they would be benefited if they could listen to more teaching conferences. Therefore, we plan to make recordings of teaching conferences in each type of clinic for use in this type of training. Since most of the nurse trainees were from health units located in predominantly rural areas, the need for emphasis of this type of health program was obvious. The Bastrop County office offers an ideal set-up for this type of health work. The organization, midwife situation, the maternity clinics, and the well child conferences make it a good rural training area. A few nurse trainees spent part of their training time in this office. During the present year it is planned to use this rural situation to better advantage.

Library facilities were inadequate during the first year of operation. Although the library facilities for the normal operation of the Travis-Bastrop County Health Unit seemed to be sufficient for the usual needs it was found that a good many books and other materials would be necessary if all of the needs of the trainees were to be met. Therefore, steps have been taken to improve and enlarge this important adjunct of any training program. A sound movie projector purchased during the first year of operation of the Training Unit has proved of great value.

Experiences gained during the school for clerks indicates the need for holding separate schools for different categories of clerks. Those working in the smaller units need to be trained in different classes from those working in larger units. Special schools for clerks handling only special types of records such as venereal disease or tuberculosis records and reports should be trained in special groups. It is planned to devote more

time in future schools of this nature to the actual clerical procedures and have available for demonstration complete representative files applicable to the different categories of clerks.

ABSTRACT OF DISCUSSION

Dr. L. P. Walter, Austin: Training of public health personnel is now an important and timely topic. Extraordinary conferences are being held throughout the country to determine means by which there may be standardization and improvement of field training for public health workers. The facts available at this time indicate that in the near future every county in the United States will be receiving the services of a full-time local health unit. This vast expansion program is necessary and desirable during peace time and such a program will be mandatory in the event of war.

I wish to emphasize that the Texas State Health Department Training Unit has been established primarily for field training and, as has well been pointed out by Dr. Primer, the public health worker can learn best by having actual experience in the field of public health.

There are now ten excellent accredited schools of public health in the United States where persons may receive didactic training. In the near future we hope that an accredited school of public health will be located in Texas.

SURGICAL TREATMENT OF INTERTROCHANTERIC FRACTURES OF THE FEMUR

FRANK C. HODGES, M. D.
ABILENE, TEXAS

The primary object of this paper is to express a firm conviction that elderly debilitated persons who sustain intertrochanteric fractures of the femur should be treated by open operation and internal fixation. Some surgeons have been advocating this for ten years but it has been only in the last year that I have come to realize that this method gives these old people their best opportunity to survive this injury and to avoid a miserable, slow death.

It is well known that the young and more physically fit will do well when treated by traction methods, but these old and debilitated persons cannot survive the prolonged recumbency that the traction methods require. It requires a much longer time for bony union to occur in these patients than in the younger ones. They frequently are incontinent of urine and many times of feces. An indwelling catheter helps but even this does not always keep them dry. They must have excellent nursing care to prevent decubital ulcers, and nursing care is now hard to get. The imposed inactivity and the pain frequently result in mental depression and even psychosis.

It is only reasonable for laymen and physicians to believe before investigation that these elderly debilitated patients cannot tolerate open reduction and internal fixation. Statistical reports from several large hospitals show that the mortality in this group treated by traction methods runs between 35 and 40 per cent and that in the same hospitals the mortality has been reduced by about 10 per cent by open reduction and internal fixation. In addition to the lowered mortality the patients are more comfortable, have fewer complications, and require less nursing and shorter hospitalization.

In a group of 34 intertrochanteric fractures that I have treated in private patients, 22 were treated by traction with a mortality of 22 per cent. Further analysis of this group of patients treated by traction revealed that 13 were over 70 years of age and that the mortality in this group was 39 per cent. It became obvious that the fatalities in the conservatively treated group were occurring principally in patients over 70 years of age. This information and statistical reports from some of the larger hospitals in this country led me to attempt open reduction and internal fixation in this older age group. Since adopting this method I have operated on 12 patients with an average age of 79 years. The mortality in this group of 12 patients treated by open reduction and internal fixation has been 33 per cent, which is 6 per cent lower than in the group of 13 who were over 70 years of age and treated by traction.

I believe that information concerning the lowered mortality following operative treatment in the older age group should be disseminated because that is the group in which it is reasonable to assume operation is not advisable unless a thorough study of the situation has been made. Some have advised that these patients be prepared for surgery over a period of days, but it has been my experience that given a patient who is not acutely ill and who has been as active as these old people ordinarily are, they do better if operated upon within a few hours of their injury. Many will require transfusion, for they are anemic and the operation will be cause for shock additional to that associated with their injury. This shock can and should be treated promptly and the operation carried out within a few hours.

Numerous devices have been designed for internal fixation and I have used many of them. Some of these are suitable in some types of fractures but not in others. I have attempted to find one device which is suitable for all of the many types of intertrochanteric fractures so that the operative procedure can be standardized to be done quick-

Read before the Section on Surgery, State Medical Association of Texas, Annual Session, Dallas, May 6, 1947.

ly. The worst of these fractures have extensive comminution over an area of about 2 inches in the intertrochanteric area. In these cases firm fixation of the internal fixation device must be had in the head of the femur and along the upper portion of the shaft of the femur with enough strength between these two points of fixation to sustain the stress of free movement in bed without breaking or bending. The Smith-Petersen nail with attachable femoral plate has met this requirement for me. The femoral plate is so made that it can be securely fixed to the nail at varying degrees to meet the angle subtended by the nail and the femoral shaft.

The treatment of intertrochanteric fractures in elderly patients is difficult and many complications can arise. They tax the ingenuity of the surgeon and the facilities of the hospital, but the plight of these patients is so pitiful I feel that all possible should be done to help them.

Complications which commonly arise are shock, infection, bending of fixation device, intrusion of the nail into the hip joint, decubital ulcers, and senile psychosis. These complications should be anticipated and prophylactic measures taken. Shock can be avoided by transfusion before surgery when indicated, by speedy operation and transfusion during or after operation when necessary. The danger of infection can be lessened by keeping the patient's hemoglobin above 80 per cent and blood proteins up, by speedy, atraumatic surgery, and by remedying any vitamin deficiency. The fixation device should be strong enough to withstand the stress to be put upon it. Intrusion of the nail may occur if the fixation device bends, permitting varus deformity. If comminution is extensive and the patient has good thigh muscles, traction may be necessary for awhile to prevent intrusion of the nail. However, the primary object in operating on these patients is to prevent the necessity of traction. To prevent decubital ulcers the patient should be kept clean and dry and should be turned frequently. The hemoglobin and blood proteins should also be kept high. Senile psychosis may be avoided by so fixing the fracture that the patient is not constantly in pain, by getting him up in a few days, and by keeping him interested in things in which he had been interested before injury.

ABSTRACT OF DISCUSSION

Dr. Felix L. Butte, Dallas: All of us who treat elderly patients with intertrochanteric fractures of the femur have experienced similar good and bad results. While I agree that some form of internal fixation is the method of choice in treating these fractures, I am still not willing to discard certain forms of traction, and particularly the well-leg traction whereby the patient can be put in a wheel chair and can be moved about relatively freely within a few days after the fracture. I do not believe any of these older patients should be treated with any of the methods of traction which confine them to bed. However, the well-leg traction treatment is still, in my opinion, acceptable in a certain percentage of these cases.

Dr. S. C. Richardson, Bryan: I think anyone that is satisfied with using as large a device as the Smith-Petersen nail in neck and intertrochanteric fractures and open reductions will find Dr. Hodges' method good in many cases.

I have used Roger-Anderson's pins and saved doing open operations on quite a number of old people with good results. Usually this procedure can be done under local anesthesia and the patient can be up in a wheel chair early with no cast being used at all.

If the two pins are put through the neck into the head at angles to each other and the outside ends of the pins are tightened together, they will hold. Then bars and pins can be used in the shaft and locked. Trouble may be encountered in getting the pins placed right, but they are small enough that they do little damage and can be removed and replaced easily until they are placed where they belong according to a roentgen-ray check.

Dr. Ben L. Schoolfield, Dallas: The essayist is to be congratulated upon his utter frankness in relating some of his poor results. It appears likely that greater progress would be achieved if more of us would tell about such results rather than boast of our good ones.

In dealing with intertrochanteric fractures of the femur, two things should be kept in mind: (1) that the physician is dealing in most cases with elderly patients and (2) that these fractures will almost invariably heal *if* the patient can be kept alive. Since these patients are on an average extremely old, rather older than the average patient with fracture of the neck of the femur, there is always the question of whether or not they will survive so much traumatism as is involved in this injury. Since the fractures will heal irrespective of the method of treatment, the important thing appears to be to make every effort at keeping the patients alive and at getting them out of the recumbent attitude as soon as possible.

Having been brought up in the Whitman school of orthopedic thinking, I began treating these fractures by the abduction method and the use of the long plaster of Paris spica. Within recent years, I have continued to use the Whitman method of reduction except that I have used the local injection of novocain with a preliminary injection of morphine. With this form of anesthesia, the physician need be in no particular hurry and there is plenty of time for roentgen-ray check-up on the reduction. Also I have been using only a short plaster spica reaching from the rib margins down to the knee of the affected side. A Steinmann pin or Kirschner wire is put through the shaft of the femur just proximal to the condyles to hold the internal rotation of the thigh and prevent any slipping up or down of the spica.

This latter procedure is accomplished with the local use of novocain and the pin or wire is incorporated in the plaster of Paris. This method entails the use of a minimal amount of anesthesia and trauma to the patient and the patient may be gotten up in a wheel chair quite early. Crutches may be used if the patient is strong enough to handle them.

Of course, some of these elderly patients will die no matter how they are treated. Some of them cannot stand that amount of injury and will die of shock or heart condition. I think the simpler the method of treatment, the more likely is the patient to survive and the method outlined above seems to meet those conditions satisfactorily. Frequent turning of

the patient while recumbent and getting the patient out of recumbency as early as conditions will allow will serve to prevent bed sores and pneumonia in most cases. Again let me insist that the greatest problem in intertrochanteric fractures of the femur is in keeping the patient alive. Nonunion of such a fracture must be a great rarity regardless of the method of treatment.

MISCELLANEOUS

COMING MEETINGS AND CLINICS

State Medical Association of Texas, San Antonio, May 2-5, 1949. Dr. Tate Miller, Dallas, President; Dr. Harold M. Williams, 700 Guadalupe St., Austin, Secretary.

American Medical Association. Interim Session, St. Louis, November 30-December 3, 1948. Dr. R. L. Sensenich, South Bend, Ind., President; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secretary.

NATIONAL AND REGIONAL

American Academy of Allergy. Dr. Will C. Sprain, New York, President; Dr. Theodore L. Squier, 424 East Wisconsin Ave., Milwaukee, Secretary.

American Academy of Dermatology and Syphilology. Dr. Clyde L. Cummer, Cleveland, President; Dr. Earl D. Osborne, 471 Delaware Ave., Buffalo, N. Y., Secretary.

American Academy of General Practice, Cincinnati, March 7-9, 1949. Dr. Paul A. Davis, Akron, Ohio, President; Dr. Mac F. Cahal, 20 N. Wacker Drive, Chicago 6, Acting Executive Secretary.

American Academy of Neurological Surgery. Dr. William S. Keith, Toronto, Canada, President; Dr. T. C. Erickson, 1300 University Ave., Madison 5, Wis., Secretary.

American Academy of Ophthalmology and Otolaryngology, Chicago, October 10-15, 1948. Dr. C. H. McCaskey, Indianapolis, President; Dr. W. L. Benedict, Mayo Clinic, Rochester, Minn., Secretary.

American Academy of Pediatrics, Atlantic City, November 20-23, 1948. Dr. John A. Toomey, Cleveland, President; Dr. C. G. Grulee, 636 Church St., Evanston, Ill., Secretary.

American Association for Thoracic Surgery. Dr. Alton Ochsner, New Orleans, President; Dr. Brian Blades, George Washington University School of Medicine, Washington, D. C., Secretary.

American Association of Genito-Urinary Surgeons. Dr. C. E. Burford, St. Louis, President; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago, Secretary.

American Association of Obstetricians, Gynecologists, and Abdominal Surgeons, Hot Springs, Va., September 9-11, 1948. Dr. Robert D. Mussey, Rochester, Minn., President; Dr. L. A. Calkins, University of Kansas Medical Center, Kansas City, 3, Secretary.

American College of Physicians, New York, March 28-April 1, 1949. Dr. Walter W. Palmer, New York, President; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secretary.

American College of Radiology, Dr. Edwin C. Ernst, St. Louis, President; Dr. Mac F. Cahal, 20 N. Wacker Drive, Chicago 6, Secretary.

American College of Surgeons, Los Angeles, October 18-22, 1948. Dr. Arthur W. Allen, Boston, President; Dr. Paul B. Magnuson, 40 E. Erie St., Chicago 11, Secretary.

American Congress of Physical Medicine, Washington, D. C., September 7-11, 1948. Dr. H. Worley Kendell, Chicago, President; Dr. Richard Kovacs, 2 E. 88th St., New York 28, Secretary.

American Dermatological Association. Dr. Harry R. Foerster, Milwaukee, President; Dr. L. A. Brunsting, 102 2nd. Ave., S. W., Rochester, Minn., Secretary.

American Gastro-Enterological Association, Atlantic City, June 3-4, 1949. Dr. A. F. R. Andresen, Brooklyn, President; Dr. Dwight L. Wilbur, 655 Sutter St., San Francisco, Secretary.

American Gynecological Society. Dr. Edmund A. Schumann, Philadelphia, President; Dr. Howard Taylor, Jr., 842 Park Ave., New York 21, Secretary.

American Hospital Association, Atlantic City, September 20-23, 1948. Mr. Graham L. Davis, Battle Creek, Mich., President; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secretary.

American Laryngological, Rhinological and Otological Society, Chicago, April, 1949. Dr. John J. Shea, Memphis, Tenn., President; Dr. C. S. Nash, 277 Alexander St., Rochester 7, N. Y., Secretary.

American Neurological Association. Dr. Stanley Cobb, Boston, President; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secretary.

American Ophthalmological Society. Dr. Henry C. Haden, Houston, President; Dr. W. S. Atkinson, 129 Clinton St., Watertown, N. Y., Secretary.

American Orthopedic Association. Dr. Robert I. Harris, Toronto, Canada, President; Dr. C. Leslie Mitchell, Henry Ford Hospital, Detroit 2, Secretary.

American Otological Society. Dr. Marvin F. Jones, New York, President; Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse 3, N. Y., Secretary.

American Pediatric Society. Dr. Grover F. Powers, New Haven, Conn., President; Dr. Henry G. Poncher, 1819 W. Polk St., Chicago 12, Secretary.

American Proctologic Society. Dr. George H. Thiele, Kansas City, Mo., President; Dr. Vernon G. Jeurink, 1612 Tremont Pl., Denver 2, Secretary.

American Psychiatric Association. Dr. Winfred Overholser, Washington, D. C., President; Dr. Leo H. Bartemeier, General Motors Bldg., Detroit, Secretary.

American Public Health Association, Boston, November 8-12, 1948. Dr. Martha M. Eliot, Washington, D. C., President; Dr. R. M. Atwater, 1790 Broadway, New York 19, Secretary.

American Roentgen Ray Society, Chicago, September 14-18, 1948. Dr. J. B. Edwards, St. Louis, President; Dr. H. Dabney Kerr, University Hospital, Iowa City, Secretary.

American Society of Anesthesiologists, St. Louis, November 4-6, 1948. Dr. Charles F. McCuskey, Los Angeles, President; Dr. Curtiss B. Hickcox, 745 Fifth Ave., New York 22, Secretary.

American Society of Clinical Pathologists, Chicago, October 10-16, 1948, Dr. Theodore J. Curphey, Hempstead, N. Y., President; Dr. A. S. Giordano, 531 N. Main St., South Bend, Ind., Secretary.

American Surgical Association. Dr. Elliott C. Cutler, Boston, President; Dr. W. M. Firor, Johns Hopkins Hospital, Baltimore 5, Secretary.

American Urological Association, Los Angeles, May 16-19, 1949. Dr. Charles C. Higgins, Cleveland, President; Dr. T. D. Moore, 899 Madison Ave., Memphis 3, Tenn., Secretary.

Association of American Physicians, Atlantic City, May 3-4, 1949. Dr. Francis G. Blake, New Haven, President; Dr. H. M. Thomas, Jr., 1201 N. Calvert St., Baltimore 2, Secretary.

Central Neuropsychiatric Association, Kansas City, October 29-30, 1948. Dr. William C. Menninger, Topeka, Kan., President; Dr. Lee Eaton, Mayo Clinic, Rochester, Minn., Secretary.

International College of Surgeons, U. S. Chapter, St. Louis, November 15-20, 1948. Dr. Herbert Acuff, Knoxville, Tenn., President; Dr. Louis J. Gariepy, 16401 Grand River Ave., Detroit 27, Secretary.

National Tuberculosis Association, Dr. James R. Reuling, Bayside, N. Y., President; Dr. H. Stuart Willis, 1790 Broadway, New York 19, Secretary.

Radiological Society of North America. Dr. L. Henry Garland, San Francisco, President; Dr. D. S. Childs, Medical Arts Bldg., Syracuse 2, N. Y., Secretary.

Southern Medical Association, Miami, Fla., October 25-28, 1948. Dr. A. LeDoux, New Orleans, President; C. P. Loranz, Empire Building, Birmingham, Ala., Secretary-Manager.

Southern Psychiatric Association. Dr. Guy F. Witt, Dallas, President; Dr. Newdigate M. Owensby, 384 Peachtree St. N. E., Atlanta, Ga., Secretary.

Southern Surgical Association, White Sulphur Springs, December 7-9, 1948. Dr. E. P. Lehmann, Charlottesville, Va., President; Dr. Alfred Blalock, Johns Hopkins Hospital, Baltimore 5, Secretary.

Southwest Allergy Forum, El Paso, April 4-5, 1949. Dr. Sim Hulsey, Fort Worth, President; Dr. O. E. Egbert, First National Bank Bldg., El Paso, Secretary.

Southwestern Medical Association, El Paso, October 28-30, 1948. Dr. Leslie M. Smith, El Paso, President; Dr. Wickliffe R. Curtis, First National Bank Building, El Paso, Secretary.

Southwest Regional Cancer Conference, Fort Worth, October 12, 1948. Secretary, 209 Medical Arts Bldg., Fort Worth.

United States-Mexico Border Public Health Association, Nogales, Ariz., and Nogales, Mexico, 1949. Dr. Victor Ocampo Alonzo, Hermosillo, Mexico, President; Dr. M. F. Haralson, 314 U. S. Court House, El Paso, Secretary.

STATE

Texas Association of Obstetricians and Gynecologists, Houston, October 15-16, 1948. Dr. Warren E. Massey, Dallas, President; Dr. George Adam, 4115 Fannin, Houston, Secretary.

Texas Chapter, American Academy of General Practice. Dr. J. B. Copeland, San Antonio, President; Dr. W. P. Higgins, Jr., Medical Arts Bldg., Fort Worth, Secretary.

Texas Chapter, American College of Chest Physicians. Dr. Robert B. Homan, El Paso, President; Dr. Charles J. Koerth, Kerrville, Secretary.

Texas Club of Internists. Dr. Julian C. Barton, San Antonio, President; Dr. Charles Barrier, Medical Arts Building, Fort Worth, Secretary.

Texas Dermatological Society, October. Dr. D. T. Gandy, Houston, President; Dr. W. Harris Connor, 601 Medical Arts Bldg., Houston, Secretary.

Texas Heart Association. Dr. Walter B. Whiting, Wichita Falls, President; Dr. Merritt B. Whitten, 1421 Medical Arts Bldg., Dallas, Secretary.

Texas Hospital Association, Galveston, April 19-21, 1949. Mr. C. J. Hollingsworth, Lubbock, President; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secretary.

Texas Neuropsychiatric Association, Waco, October 25, 1948. Dr. M. J. Cooper, San Antonio, President; Dr. David Wade, 604 Capital National Bank Bldg., Austin, Secretary.

Texas Orthopedic Association, San Antonio, May 2, 1949. Dr. Ruth Jackson, Dallas, President; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secretary.

Texas Pediatric Society, Dallas, October 15-16, 1948. Dr. C. B. Alexander, San Antonio, President; Dr. John E. Ashby, 3610 Fairmount, Dallas, Secretary.

Texas Public Health Association, San Antonio, February, 1949. Dr. Austin E. Hill, San Antonio, President; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secretary.

Texas Radiological Society, Fort Worth, January 7-8, 1949. Dr. L. M. Garrett, Corpus Christi, President; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secretary.

Texas Railway and Traumatic Surgical Association, San Antonio, May 2, 1949. Dr. Denman C. Hucherson, Houston, President; Dr. W. F. Parsons, First National Bank Bldg., Fort Worth, Secretary.

Texas Rheumatism Association. Dr. Howard C. Coggeshall, Dallas, President; Dr. J. Paul Thomas, Medical Arts Bldg., Dallas, Secretary.

Texas Society for Mental Hygiene, Dallas, March 3-5, 1949. Dr. Ozro T. Woods, Dallas, President; Mrs. Elizabeth F. Gardner, 1617 Watchhill Road, Austin 21, Executive Secretary.

Texas Society of Anesthesiologists. Dr. J. C. Youngblood, Houston, President; Dr. H. C. Slocum, 928 Strand, Galveston, Secretary.

Texas Society of Gastroenterologists and Proctologists, San Antonio, May 2, 1949. Dr. Alvin Baldwin, Dallas, President; Dr. Carl Giesecke, 1602 Nix Professional Bldg., San Antonio, Secretary.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, December 3-4, 1948. Dr. S. N. Key, Sr., Austin, President; Dr. John L. Matthews, 929 Nix Professional Bldg., San Antonio, Secretary.

Texas Society of Pathologists, Dallas, January 30, 1949. Dr. W. W. Coulter, Sr., Houston, President; Dr. C. T. Ashworth, 604 Medical Arts Bldg., Fort Worth, Secretary.

Texas State Urological Society, Temple. Dr. Michael K. O'Heeron, Houston, President; Dr. E. O. Bradfield, Scott and White Clinic, Temple, Secretary.

Texas Surgical Society, Fort Worth, October 4-5, 1948. Dr. G. W. N. Eggers, Galveston, President; Dr. Truman G. Blocker, University of Texas Medical Branch, Galveston, Secretary.

Texas Tuberculosis Association, San Angelo, September 24-25, 1948. F. K. Dougharty, Liberty, President; Miss Pansy Nichols, 700 Brazos, Austin, Executive Secretary.

DISTRICT

Second District Society, Lubbock, October 5-6, 1948. Dr. R. B. G. Cowper, Big Spring, President; Dr. Carl Uthoff, Big Spring, Secretary.

Third District Society, Lubbock, October 5-6, 1948. Dr. C. E. High, Pampa, President; Dr. Kenneth Flamm, Amarillo, Secretary.

Fourth District Society, Ballinger, October 12, 1948. Dr. J. C. Young, Coleman, President; Dr. Charles F. Bailey, Ballinger, Secretary.

Fifth and Sixth Districts Society, Corpus Christi, July, 1949. Dr. D. R. Knapp, Kerrville, President; Dr. Charles Tennison, Nix Professional Building, San Antonio, Secretary.

Seventh District Society. Dr. David Wade, Austin, President; Dr. Joe W. Bailey, 1411 San Antonio St., Austin, Secretary.

Eighth, Ninth and Tenth Districts Society. Dr. T. O. Woolley, Orange, President; Dr. James Greenwood, Jr., 518 Medical Arts Bldg., Houston 2, Secretary.

Eleventh District Society, Tyler, October 22, 1948. Dr. Harvey Bell, Palestine, President; Dr. C. B. Young, 929 S. Confederate, Tyler, Secretary.

Twelfth District Society. Dr. R. R. White, Temple, President; Dr. H. F. Connally, Jr., Amicable Bldg., Waco, Secretary.

Thirteenth District Society, Mineral Wells, November 11-12, 1948. Dr. Fred Harrell, Olney, President; Dr. S. W. Wilson, Medical Arts Bldg., Fort Worth, Secretary.

Fourteenth District Society, Ennis, January, 1949. Dr. W. I. Southerland, Sherman, President; Dr. John Bagwell, Medical Arts Building, Dallas, Secretary.

Fifteenth District Society, Daingerfield, October 19, 1948. Dr. W. S. Terry, Jefferson, President; Dr. James Harris, Marshall, Secretary.

CLINICS

Dallas Southern Clinical Society, Dallas, March 14-17, 1949. Miss Thelma J. Webb, Medical Arts Bldg., Dallas 1, Executive Secretary.

International Post-Graduate Medical Assembly of Southwest Texas, San Antonio, January 25-27, 1949. Dr. John J. Hinchey, 643 Moore Bldg., San Antonio 5, Secretary.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, October 20, 1948. Dr. Charles H. Brown, Wichita Falls, Program Chairman.

Oklahoma City Clinical Society Conference, Oklahoma City, October 25-28, 1948. Muriel R. Waller, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secretary.

Post Graduate Medical Assembly of South Texas, Houston, December 7-9, 1948. Dr. E. Trowbridge Wolf, 229 Medical Arts Bldg., Houston, Secretary.

TEXAS DIABETES ASSOCIATION

The Texas Diabetes Association is being organized at the suggestion of the American Diabetes Association and has already had one meeting. Some twenty physicians from throughout the state gathered in Gainesville on July 17 to hear a lecture by Dr. Arthur R. Colwell, Chicago specialist in diabetes, and to make plans for a permanent organization in Texas. Dr. J. Shirley Sweeney, Gainesville, was elected president; Dr. Ben F. Smith, Houston, first vice-president; Dr. Haynes Harvill, Dallas, second vice-president; Dr. James H. Harris, Marshall, secretary; and Dr. W. N. Powell, Temple, treasurer.

A charter has been filed, and a board of directors is being chosen. Dr. Charles H. Best, University of Toronto, Canada, co-discoverer of insulin and president of the American Diabetes Association, will serve as honorary chairman of the board. Dr. Smith is chairman of a committee to draw up a constitution and by-laws.

In addition to the clinical society, the organizers of the Texas Diabetes Association plan to have a lay council to disseminate knowledge about diabetes, and laymen from various parts of the state will be represented on the board of directors.

DALLAS HEALTH MUSEUM CELEBRATES BIRTHDAY WITH EXPANDED PROGRAM

The second anniversary of the Dallas Health Museum will be observed October 9 when the State Fair of Texas opens. During its two years of existence, the museum has greatly increased its program of activities, many of which are described in the first issue of a quarterly bulletin, *Dallas Health Museum*, which was distributed in August.

Of major interest as the museum closes its second year of making "nontechnical health information interesting and readily available to the layman" is the fact that the major portion of the internationally famous Oberlander collection of health education exhibits is being loaned to it for display. More than fifty exhibits, including the "Transparent Man" and an 8 foot ear demonstrating how fluid in the canals of the organ control the sense of balance, are included in the loan. They are part of the collection which the Oberlander Trust and Foundation, Philadelphia, assembled for display at the New York World's Fair by the American Museum of Health, New York City. Mr. Homer Calver, director of the American Museum, arranged for transfer of the exhibits to Dallas from the Cleveland Health Museum, where they have been for the past several years. The transfer is part of a cooperative plan worked out among the nation's health museums for the exchange of educational material.

Necessary refurbishing of the exhibit is being done in the newly established Health Museum workshop, which is equipped with a variety of tools for production of new exhibits as well as for repairing and renovating older displays. A seven unit display on dental science constructed in the workshop from designs by Vic Klein, museum industrial design consultant, with the Dallas County Dental Society cooperating will be dedicated October 5.

A summer activity of the museum has been twice-weekly arts and crafts classes for children in which two art instructors have led the children in illustrating certain health topics in clay, drawings, and other media.

Courses in parentcraft for future mothers and fathers, well child conferences, free chest x-ray ex-

aminations, a question box on health subjects, and daily guided tours are other services of the museum. Thirty-four college and school groups have used the Health Museum facilities as the basis for field trips and study periods since January, 1948.

In April the first annual Dallas Health Museum Award for Outstanding Contribution to Public Health in Texas was presented to Dr. Robert L. Sutherland, Austin, director of the Hogg Foundation for Mental Hygiene.

The work of the Dallas Health Museum has already gained international recognition. An invitation was recently received by museum officials to present a paper at the Scientific Museum Section of the International Organization of Museums Congress in Paris. The congress is part of the United Nations Educational, Social, and Cultural Organization. Although unable to attend, Mrs. Marguerite Hays, director of the museum, prepared a report on the Dallas Health Museum to be read at the congress.

UNIVERSITY OF TEXAS POSTGRADUATE COURSES

The 1948-1949 schedule of postgraduate refresher courses to be offered by the University of Texas School of Medicine, Galveston, has been released by Dr. T. G. Blocker Jr., director of the Postgraduate Division. The courses and the dates for which they will be offered are as follows:

Electrocardiography, Friday afternoons, October-December 17 (individual arrangement).
Tumor Conference (fall), October 25-28.
Psychiatry, November 1-6.
Pediatrics (fall), November 8-13.
Physical Medicine, March 7-11 (tentative).
Obstetrics, March 21-26.
Pediatrics (spring), April 11-16.
Tumor Conference (spring), May 4-7.

The tumor conference in October will be devoted to tumors of the lymphoid system, tumors of the thyroid, and intrathoracic tumors. This course is the fourth of a series of courses dealing with various types of tumors, planned to run over a period of three years.

SAN ANTONIO TUMOR SEMINAR SCHEDULED

Dr. Shields Warren, Boston, will conduct the fifth annual tumor seminar in San Antonio on October 23 in the auditorium of the fourth annex of Brooke General Hospital. The meeting will begin at 9:30 a. m. "Diseases of the Reticulo-Endothelial System" is the subject chosen for study this year, and 25 cases will be discussed by Dr. Warren.

Physicians interested in attending the seminar should communicate with Dr. B. F. Stout, Medical Arts Building, San Antonio. Visitors need not bring their own microscopes.

Luncheon will be served at the hospital, and a dinner will be held at 7:30 p. m. at La Villita for guests and their wives.

TEXAS TUBERCULOSIS ASSOCIATION

The thirty-eighth annual meeting of the Texas Tuberculosis Association, together with meetings of the Texas Trudeau Society and the Texas Conference of Tuberculosis Secretaries, will be held at the St. Angelus Hotel, San Angelo on September 24 and 25. Physicians of the state have been cordially invited to attend, and scientific presentations of particular interest to them have been planned.

Dr. J. B. McKnight, superintendent of the State Tuberculosis Sanatorium near San Angelo, will entertain visitors with a barbecue at 6 p. m., September 23. Various departments of the Sanatorium will be open to visitors before the barbecue.

ATABRINE EFFECTIVE IN TULAREMIA

Atabrine, the principal reliance for malaria control in the Pacific and Mediterranean theaters during the war, may be effective in controlling undulant fever and tularemia. Writing in the June, 1948, issue of *The Illinois Medical Journal*, D. H. Ecke, M.D., of Vandalia, and Dean H. Ecke of the Illinois History Survey, Urbana, described 8 cases of tularemia and 4 cases of undulant fever which Dr. Ecke successfully treated with atabrine.

The tularemia cases included in the report were all treated up to April, 1948. "Without exception, atabrine produced a favorable response within one to three days and not one case required further treatment after the fifth day," the report states. "In none of these cases has there been a recurrence of symptoms."

Dr. Ecke employed the same treatment in 4 undulant fever cases. All symptoms of undulant fever disappeared within five days after atabrine was started. One patient relapsed after almost five years, but recovered after two or three days of atabrine treatment.

INTERNSHIPS IN VA HOSPITALS

A limited number of internships in Veterans Administration hospitals will be offered qualified medical graduates, the Veterans Administration has announced. The internships will be available for "straight" subjects in surgery, general medicine, and other fields and for "rotating" service in surgery, general medicine, and related subjects, including obstetrics and pediatrics, provision for which will be made in other than Veterans Administration hospitals.

Internships will be for one year, subject to reappointment for one year, and will be available only in the seventy-five Veterans Administration hospitals now offering residency training programs in cooperation with class A medical schools.

In Texas, internships will be offered at the Veterans Administration hospitals in Dallas, Legion, McKinney, Temple, and Waco. Residency training is also offered at the mental hygiene clinic in the regional office of the Veterans Administration, Love Field, Dallas.

Announcement has also been made that the Veterans Administration has immediate openings for at least one hundred physicians interested in taking residency training in psychiatry or neurology or both. Applications should be made to the Chief Medical Director, Veterans Administration, Washington 25, D. C.

LIBRARY NOTES

PACKAGE SERVICE

The package library consists of collections of reprints and other periodical material on various subjects, prepared for lending to members of the Association. Requests for packages should be addressed "Library, State Medical Association of Texas, 700 Guadalupe Street, Austin, Texas." Twenty-five cents in stamps should be enclosed with the request to cover postage and part of the expense of collecting the material. Packages are allowed to remain in the hands of the borrower for 14 days.

ACCESSIONS

The following additions were made to the Library during August:

Reprints received, 436.

Journals received, 179.

Books received, 13.

General Endocrinology, Turner, from W. B. Saunders, Philadelphia.

Morphologic Hematology and *The Spleen and Hypersplenism*, Dameshek (editor); *Hemolysis and Related Phenomena*, Ponder; *Venous Thrombosis and Pulmonary Embolism*, Neuhof; and *Biology of Disease*, Moschcowitz, from Grune & Stratton, New York.

Practical Therapeutics, Rehfuss and others; *Manual of Leprosy*, Muir; and *Bacterial and Virus Diseases*, Parish, from Williams & Wilkins Company, Baltimore.

Human Breeding Survival, Burch and others, from Penguin Book Incorporation, New York.

More than Armies, Mooney, from Mathis, Van Nort and Company, Dallas.

The Technic and the Art of Medical Writing, Fishbein, from Blakiston Company, Philadelphia.

Physiology of Man in the Desert, Adolph, from Interscience Publishers, Inc., New York.

SUMMARY OF SERVICE

Local users, 39.	Borrowers by mail, 28.
Items consulted, 581.	Packages mailed, 32.
Items taken out, 174.	Items mailed, 323.

Total number of items consulted and loaned, 1,078.

MOTION PICTURE FILM LIBRARY

Motion picture films on medical subjects, 16 mm., both silent and sound, some in color, and suitable for either medical or lay audiences, are available for loan to county medical societies, hospital staffs, or individual physicians, on request. Borrowers will be required to pay only the cost of shipment of the films, by express, with insurance, and for any damage to films while in the hands of the borrower.

Requests for films should be addressed to "Motion Picture Film Library, State Medical Association of Texas, 700 Guadalupe Street, Austin, Texas." A list of available films, with descriptions, will be furnished on request.

The following motion picture films were loaned by the Film Library during August:

Acute Appendicitis in Childhood (Mead Johnson)—All Saints Hospital Resident Staff, Fort Worth.

Animated Hematology (Armour Laboratories)—Coffey Clinic, Fort Worth.

As Others See Us (American Hospital Association)—Coffey Clinic, Fort Worth.

Bleeding Tendency (Mead Johnson)—Dr. T. L. LaMotte, Harlingen.

Cervical Smear II (Dr. K. J. Karnaky)—Gilbert and Roberts Clinic, Irving.

Cloud in the Sky (Spanish) (Texas Tuberculosis Association)—Junior Service League Well Baby Clinic, Brownsville.

Conquering Darkness (Hurst Clinic)—Dr. T. J. LaMotte, Harlingen.

Human Fertility (Ortho-Products Inc.)—Dr. T. J. LaMotte, Harlingen.

Nasal Sinusitis (E. Fougera & Co.)—Dr. Johnnie Monaghan, Jr., Fort Worth.

Parkinsonism (Lederle Laboratories)—Dr. Johnnie Monaghan, Jr., Fort Worth.

Safer Gastrectomy (Billy Burke Productions)—Coffey Clinic, Fort Worth.

Spontaneous Delivery (Mead Johnson)—Dr. Johnnie Monaghan, Jr., Fort Worth.

Time Is Life (American Cancer Society)—Gilbert and Roberts Clinic, Irving.

Traitor Within (American Cancer Society)—Gilbert and Roberts Clinic, Irving.

Trichomonas Vaginalis (Dr. K. J. Karnaky)—Gilbert and Roberts Clinic, Irving.

When Bobby Goes to School (Mead Johnson)—Well Baby Clinic, Brownsville.

BOOK REVIEWS

[1]**Textbook of General Surgery.** By Warren H. Cole, M.D., F.A.C.S., and Robert Elman, M.D., F.A.C.S. Fifth edition. Cloth, 1,160 pages. Price, $11. New York and London, D. Appleton-Century Company, 1948.

This latest edition of an excellent textbook of surgery contains many new features which make it even more desirable as a reference book for the general practitioner and the surgical specialist. New chapters on preoperative and postoperative care and surgical diseases of the chest, as well as additions to the chapters on chemotherapy, further increase the value of this book. It is highly recommended to anyone interested in surgery and the surgical specialties.

[2]**Advances in Military Medicine.** By American Investigators Working under the Sponsorship of the Committee on Medical Research. Cloth, 882 pages, illustrated, two volumes. Price $12.50. Boston, Atlantic Monthly Press, Little, Brown and Company, 1948.

These two books make up an official record of medical progress during the war. It is a record of achievement by medical men and their associates in research, the tremendous effects of which are now beginning to be felt throughout the fields of medicine and public health.

Specialists in various fields of study have contributed to the information gathered in these sections: medicine, surgery, aviation medicine, physiology, chemical-warfare agents, anti-pest agents, adrenocortical steroids, malaria, penicillin, and sensory devices.

With the worldwide scope of the war necessitating troops scattered in areas from the equator to the poles, and with doctors, scientists, and technicians at work with unlimited funds furnished by the government, progress and developments of almost unbelievable proportions have been reported. The information gathered together in these two books would usually be found in an unlimited number of books.

"Advances in Military Medicine" may well be recommended as a ready reference to such perplexing problems as tropical diseases, repair of peripheral nerve lesions, motion sickness, shock, blood substitutes, history of development of DDT, malaria, penicillin, and sensory devices. These represent just a few of the many interesting subjects discussed.

[3]**Laboratory Technique in Biology and Medicine.** By E. V. Cowdry, Professor of Anatomy, Washington University, and Director of Research, Barnard Free Skin and Cancer Hospital, St. Louis. Second edition. Cloth, 269 pages. Price, $4. Baltimore, Williams and Wilkins Company, 1948.

Dr. Cowdry's second edition of "Laboratory Technique in Biology and Medicine" presents to physicians an excellent reference book relative to problems in biology. The content is basic and the result of extensive experience within the field of investigation. The alphabetical order of subject matter permits quick, orderly reference to the normal, relative normal, or problem status, and thereafter guidance is given with named procedures, materials, and indicated reference.

[1]Reviewed by Robert D. Bickel, M.D., Fort Worth.
[2]Reviewed by J. H. Steger, M.D., Fort Worth.
[3]Reviewed by H. B. Williford, M.D., Beaumont.

It is not wise to attempt listing of unit interest, because the reader will most often carry a problem to the book. However, many individualistic and interesting enumerations will initiate scientific problems. The book is thought-provoking, non-enigmatical, though purposely expansile.

[4]**Clinical Diagnosis By Laboratory Methods.** By James Campbell Todd, Ph.D., M. D., and Arthur Hawley Sanford, A.M., M.D. Eleventh edition. Cloth, 954 pages. Price, $7.50. Philadelphia and London, W. B. Saunders Company, 1948.

The eleventh edition of this well known book has been extensively revised. New illustrations have been added, and many of the older figures have been redrawn. A new feature is the listing of references at the end of each chapter.

In the section on urine, Sulkowitch's test for calcium, the methods of Jacobson and Milner for the identification of Bence-Jones protein, and Sumner's quantitative test for dextrose have been added. In the section on blood, the discussion on coagulation has been revised, the Ivy method for bleeding time has been added, the Rh factor is more fully discussed, and new material on blood dyscrasias has been inserted.

Under gastric analysis, the method of Foley and Andosca for the examination of gastric contents for Mycobacterium tuberculosis is included. No details are given for the microscopic examination of bile and duodenal contents.

The chapter on clinical chemistry contains new material and methods, and the order of presentation has been changed. Watson's method for the quantitative determination of urobilinogen is briefly discussed in the chapter on feces. Under puncture fluids a brief discussion of the smear methods of cytodiagnosis is given, but the technique of Papanicolaou is not mentioned. A separate chapter is now devoted to viruses and rickettsias, while a chapter on mycology has been revised. Biologic skin tests are well covered in a separate chapter.

No detailed techniques are given for the determination of any vitamins except cevitamic acid. Hormones are briefly covered, with the frog test for pregnancy included.

The quality of the paper and print is much superior to that of the previous edition. The book maintains its former high standard and can be strongly recommended to students, practitioners, and laboratory workers.

[5]**Neuroanatomy.** By Fred A. Mettler, A. M., M. D., Ph.D., Associate Professor of Anatomy, College of Physicians and Surgeons, Columbia University, New York. Second Edition. Cloth, 536 pages. Price, $10. St. Louis, C. V. Mosby Company, 1948.

The second edition of Dr. Mettler's "Neuroanatomy" has brought this subject up to date. It is not, as are many second editions of books, just another reprint. The author gives a complete and accurate description of the blood supply and venous drainage of the central nervous system. As he states in the preface, much of the new material added is not easily available in English. This section serves well to show the relationship of the blood supply to disease and pathology of the central nervous system.

The discussion of the connections of the subdivisions of the thalamus with the higher portions of the brain is enlightening and helps to clear up the conception of the functions of these deeper-lying portions of the brain.

The author's concept of the connections of the basal ganglia is especially interesting in view of the fact that it suggests a clearer idea of the function of these not too well understood portions of the brain.

As stated in a review of the first edition of this book, it might be hoped that the author will see fit to include in future editions a somewhat fuller discussion of the functional side of the nervous system. This text contains excellent illustrations, and the descriptive material is good and concise. It can be recommended to both students and graduate physicians.

[6]**Heart—A Physiologic and Clinical Study of Cardiovascular Disease.** Edited by Alde A. Luisida, M. D., Instructor in Physiology and Pharmacology, Tufts Medical School; Lecturer in Medicine and Lecturer Post Graduate Division, Tufts Medical School; Associate in Medicine, Beth Israel Hospital, Boston; Former Professor of Medicine, Ferrara, Italy. With a foreword by Herman L. Blumgart. Cloth, 653 pages. Price, $10. Baltimore, Williams and Wilkins Company, 1948.

Although a faithful and silent organ in health, the heart in disease becomes a difficult and complex problem. The author realized this situation and therefore undertook to cover the multiplicity of cardiac derangements by first establishing the known anatomic and physiologic facts of a normal heart before digressing to the great variety of pathologic conditions.

To enable the reader to have a vivid picture of each cardiac aberration, special effort has been taken to supplement the discussion by visual means. This is accomplished by actual photographs, diagrams, drawings, and tracings. It is true that if a person reads the text as a novel, the repetition of these illustrations becomes tiresome, but if he uses the text for reference work, a composite picture of the particular cardiac condition to be studied is complete in each detail. There is no excess statistical data to divert attention from the central theme. Instead, the reference work is marked by a heavy type numeral in parenthesis, and the complete bibliography is listed at the end of each chapter.

The text is written in simple terms and has an excellent arrangement. All related conditions are included in a single chapter. For instance, in the chapter on "Disease of the Myocardium" are found all those conditions that have to do with changes in structure and function of the heart muscle. Then each individual condition is further broken down into etiology, symptoms, signs, tracings, and differential diagnosis. This gives a complete discussion of a particular phase without any great amount of confusion.

Unquestionably, this text is an asset in the field of cardiology, and is the outgrowth of intensive study and experience. Such material, which has been gained through many fruitful years, should not be lost, but transmitted to those who are especially interested in the heart as it functions in health and disease.

[7]**The Years After Fifty.** By Wingate M. Johnson, M. D., Professor of Clinical Medicine and Chief of Private Diagnostic Clinic of Bowman Gray School of Medicine of Wake Forest College. Cloth, 153 pages. Price, $2. New York and London, McGraw-Hill Book Company, Inc., 1948.

Although this book is written primarily for the

[4]Reviewed by W. N. Powell, M.D., Temple.
[5]Reviewed by Dr. W. W. Looney, M.D., Dallas.
[6]Reviewed by I. H. Readinger, M.D., Fort Worth.
[7]Reviewed by James P. Bridges, M.D., Abilene.

layman, it makes excellent reading for physicians. Dr. Johnson, the author, is one of the leading champions of the "family doctor," and he presents his subject with the understanding and wisdom of an observant family physician. Comprehensive in its scope, brief in its presentation, this book contains informative reading on how to "grow old gracefully." Frequent quotations from ageless literature are given, ranging from Plato through Stieglitz.

This book presents to the reader impressions of what medicine can now offer to promote the comfort and usefulness of persons living beyond the age of 60. It makes interesting and profitable reading for the person approaching this age with trepidation and concern.

[5]**Psychology and Psychiatry.** By Wendell Muncie, M. D. Second edition. Cloth, 609 pages. Price, $9. St. Louis, C. V. Mosby Company, 1948.

This book by Dr. Muncie should be a "must" for all psychiatrists. It re-orients in a clear fashion the various approaches to psychiatric idealogy. The section on "Evaluation of the Personality" is complete and valuable to the student. The examination section is extremely complete and perhaps a bit too detailed for the average reader, but excellent for the advanced student. Probably one of the most valuable features of the entire book is the presentation of the histories manifesting various types of reactions. They are fine and represent an attitude of real study of the human being rather than an attempt at didactic diagnosis.

Any person interested in psychiatry, whether a medical student, general practitioner, teaching psychiatrist, or in private practice, whether intramural or extramural, will find in this volume an enlightening and thought provoking stimulus.

Diseases of Metabolism. Edited by Garfield G. Duncan, M.D. Second edition. Cloth, 1,000 pages, illustrated. Price, $12. Philadelphia, W. B. Saunders Company, 1947.

This volume is a 1,000 page work edited by Dr. Duncan, who writes the introductory chapter on "General Metabolic Considerations" and a complete review of those conditions which alter the basal metabolic rate.

Each chapter is a contribution by some authority on the specific type of metabolism under discussion. For instance, the chapter on "Carbohydrate Metabolism" is written by Dr. C. N. H. Long, Sterling professor of physiological chemistry, Yale University School of Medicine. He discusses the importance of carbohydrate, the digestion, absorption, and fate of carbohydrate in the body, and the part played by the liver skeletal muscles, anterior pituitary, and so forth in the process.

Protein metabolism is well and ably handled by Abraham White, associate professor of physiological chemistry, Yale University School of Medicine. Tom P. Spies and Hugh R. Butt discuss vitamins and avitaminosis and the part these elements play in disease processes affecting metabolism.

Thus, each chapter deals with some phase of metabolism and usually starts with a brief historical and experimental background with a fair presentation of pros and cons, and ends with a grouping and description of the disease phase of differential diagnosis and treatment of the disease. It is a "must" for the general practitioner or the specialist who wishes to review experimental work of the laboratory worker as correlated with clinical medicine.

[5]Reviewed by Charles H. Brown, M.D., Wichita Falls.

NEWS

Contributions to this department will be appreciated. News should be of general medical interest, such as public health activities, new hospitals, personal items of more than local value, etc. News for a particular number of the JOURNAL should be in the hands of the Editor not later than the fifteenth of the preceding month.

Meetings of Interest in the near future include the following:

Hospital Standardization Conference, Los Angeles, October 18-22. This annual conference will be held in conjunction with the clinical congress of the American College of Surgeons and will include lectures and discussions of interest to all types of personnel concerned with hospitals, including physicians and surgeons. Additional information may be secured from the College, 40 E. Erie Street, Chicago.

Southern Medical Association, Miami, Fla., October 25-28. Twenty-one section meetings, two general sessions, one conjoint meeting with the American College of Chest Physicians, Southern Chapter, and "Miami Day" general clinical sessions are scheduled. Hotel reservations will be handled by the Hotel Committee, Southern Medical Association Meeting, c/o City of Miami Convention Bureau, 320 N. E. Fifth Street, Miami 32, Fla.

Oklahoma City Clinical Society Conference, Oklahoma City, October 25-28. Seventeen distinguished guests will lecture; postgraduate symposia and round-table luncheons will be held; a clinical-pathological conference has been arranged; and entertainment is being planned. Additional information may be secured from the Executive Secretary, 512 Medical Arts Building, Oklahoma City.

National Society for Crippled Children and Adults, Chicago, November 15-17. Facts on progress in work with the handicapped will be presented by speakers in the fields of medicine, health, and education. Details of the meeting are available at Society headquarters, 11 S. LaSalle, Chicago.

The Texas Physicians Committee met in Dallas on August 8 to hear discussions of the history and plans of the National Physicians Committee, with which the Texas group is affiliated. Arthur L. Conrad, Chicago, associate administrator, and Dr. Edward H. Cary, Dallas, chairman of the National Physicians Committee, were the principal speakers. Dr. F. J. L. Blasingame, Wharton, was reelected chairman of the Texas organization; Dr. R. W. Kimbro, Cleburne, was elected vice-president; and Dr. H. E. Griffin, Graham, was elected secretary-treasurer.

The College of American Pathologists sponsored a regional meeting in Dallas, September 6-7. Dr. A. E. Mourant of the Blood Grouping Laboratory, Lister Institute, London, England, and Dr. Paul A. Owren, University of Oslo, Oslo, Norway, were the guest speakers for a program which emphasized blood studies. Radioactive isotopes and the effects of atomic radiation on body tissues were also considered.

The National Medical Association, meeting in New York in August, elected Dr. C. A. Whittier, San Antonio, as president. Dr. Whittier, a general physician and surgeon, is a past president of the Lone Star State Medical Association and has been prominent in other activities of the Negro medical profession of Texas.

An Ambulance Plane Is Available to physicians of North Texas, according to the Wichita Falls *Daily Times*. The Alexander Clinic of Wichita Falls has recently secured a completely equipped ambulance plane, which will be placed at the disposal of physicians at nominal cost in case of emergency. Dr. Ted Alexander, director of the clinic, and his associate,

Dr. Charles Hawes, are both licensed pilots, and the services of Jack Holman, another pilot, are available if needed. The plane has room for a patient, a nurse, and a physician.

Reorganization of the Gonzales Warm Springs Foundation is well under way with formation of a new medical staff and adoption of by-laws, reports the San Antonio *Express*. Dr. Peter M. Keating, San Antonio, has been named resident medical director and new equipment is being added to the facilities of the 40 acre tract set aside for the care of poliomyelitis patients. It is hoped the foundation will soon secure the approval of the American College of Surgeons. The medical staff elected Drs. David M. Shelby, Gonzales, president; Alfred I. Heinen, Seguin, vice-president; Sidney Stovall, San Antonio, secretary; Philip Magrish, San Antonio, chief of the medical division; Walter Stuck, San Antonio, chief of surgical service; and C. B. Lynn, Austin, chief of dental staff.

Texas Medical Center.—Two committees to aid in the integration of the Texas Medical Center program have been appointed, according to the Houston *Chronicle*. A professional committee to act on matters pertaining to the teaching responsibilities of the center and cooperating units is headed temporarily by Dr. James A. Greene as chairman and by Dr. Russell J. Blattner as secretary. Both men are on the staff of Baylor University College of Medicine. The institutional committee, which will handle matters of common interest among the units in the center, has as temporary chairman James W. Rockwell, a regent of the University of Texas, and as temporary secretary Mrs. Elna Birath, bursar of the University of Texas Dental School.

Plans for the University of Texas School of Public Health, authorized by the State Legislature as one of the participating units of the Texas Medical Center, were discussed in Houston in August, reports the Houston *Chronicle*. Dr. R. E. Dyer, Bethesda, Md., director of the National Institute of Health; Dr. T. S. Painter, Austin, president of the University of Texas; Dr. E. W. Bertner, Houston, president of the Texas Medical Center; Dr. Chauncey D. Leake, Galveston, vice-president and dean of the University of Texas Medical Branch; and Dr. R. Lee Clark, Jr., Houston, director of the M. D. Anderson Hospital for Cancer Research, conferred. It is planned to request the next Legislature to appropriate funds for the School of Public Health, which will be the ninth such school in the United States.

A survey of children's hospitals throughout the United States, Mexico, and Canada is being undertaken by the Texas Children's Foundation preparatory to erection of a 200 bed children's hospital in the Texas Medical Center, according to the Houston *Chronicle*. Dr. Russell J. Blattner, chairman of the Department of Pediatrics at Baylor University College of Medicine, and Milton Foy Martin, Houston architect, are on a tour to inspect hospital facilities. Money for the survey has been made available by proceeds from the Houston Horse Show.

The new 400 bed Hermann Hospital building, due to open about November 1, will include an expanded physical medicine department which will be one of the largest in the country, the Houston *Chronicle* informs. Facilities in the new department will care for more than seven times the patients now treated. Classes in the school of physical therapy, established last year at the hospital, will begin October 1.

The M. D. Anderson Hospital for Cancer Research has appointed four new staff members, the Houston *Chronicle* states. Dr. Charles C. Henderson, Dallas, has been named a fellow in clinical research; Dr. Frank S. Rose, former chief of surgical services at Randolph Field, has been made a resident in surgery; Alton Landua, Houston, and O. K. Towler, Baytown, have been named to the research laboratory staff.

Baylor University College of Medicine has received a $50,000 gift for scholarships in pediatrics from Houston Endowment, Inc., the philanthropic corporation established by Mr. and Mrs. Jesse Jones, reports the Houston *Post*. To be known as the Jones Pediatric Scholarships (Jesse Jones scholarships for men, Mary Gibbs Jones scholarships for women), the gifts will provide from $400 to $800 a year for from three to six years to undergraduates and graduates who wish to specialize in child health. Baylor has also announced an affiliation agreement with the Children's Medical Center of Boston to provide for an exchange of students for pediatric study.

A laboratory of clinical electrophysiology will begin operation at Baylor University College of Medicine about the first of the year, the Houston *Chronicle* informs, with Dr. Peter E. Kellaway, associate professor of physiology and neurology, as director. The Baylor laboratory will do pioneering work in the field of electrical phenomena produced through normal body functions and their application in the study of poliomyelitis, the heart, brain, and muscles.

Dr. Michael E. DeBakey, associate professor of surgery at Tulane University, has been named Judson L. Taylor professor of surgery at Baylor University College of Medicine, according to the Houston *Press*.

Southwestern Medical College with the opening of its school year is undertaking an intensified cancer program centered in its new biophysics laboratory directed by Dr. Allen Reid, head of the Department of Biophysics. Funds for cancer work totaling more than $150,000 have been made available to the college since its founding.

Dr. Herman I. Kantor, clinical assistant professor of obstetrics and gynecology at Southwestern Medical College, has been awarded the annual prize of the Central Association of Obstetricians and Gynecologists for the "best investigative work done during the past year." Dr. Kantor appeared at the annual meeting of the association in September to report on his study concerning the urinary bladder during labor.

Dr. Thomas W. Farmer, former instructor in internal medicine at Johns Hopkins University, has been named assistant professor of neurology at Southwestern Medical College, the Dallas *Times-Herald* reports.

The University of Texas Medical Branch has announced the appointment of Dr. Wendell H. Griffith, professor of biochemistry, St. Louis University Medical School, as professor of biochemistry and nutrition at the Medical Branch and of Col. Roy Stout, USA (Ret.), an authority on plastic and maxillo-facial surgery, as consultant dentist.

Dr. Marshall Brucer, assistant professor of physiology at the University of Texas Medical Branch, has been appointed chairman of the board of medical consultants for research studies involving radioactive isotopes in treatment of cancer at the Oak Ridge Institute of Nuclear Studies, reports the Galveston *Tribune*.

Dr. Edgar J. Poth, professor of surgery, has received a grant of $3,000 from the Upjohn Company, Kalamazoo, Mich., for a special study on the action of antibiotics in promoting healing in surgical wounds. Dr. Poth reported some of the results of his studies at the Pan-Pacific Surgical Congress in Honolulu, August 30-September 10.

Friends of the late Dr. Albert O. Singleton, professor of surgery at the Medical Branch, have placed a bronze plaque bearing a bas-relief of Dr. Single-

ton near the entrance to the special surgical unit of John Sealy Hospital, recently completed as a result of plans made by Dr. Singleton.

Personals

Dr. McIver Furman, Corpus Christi, was named representative director of the National Tuberculosis Association for a two year term at its annual meeting in June in New York, according to the *Bulletin* of the Association. *Dr. James E. Dailey*, Houston, was elected to the advisory board of the American Trudeau Society, and *Miss Emmeline J. Renis*, executive secretary of the Houston Anti-Tuberculosis League, was made president-elect of the National Conference of Tuberculosis Secretaries.

Drs. George E. Clark, Austin; *Olin B. Gober* and *J. G. Rodarte*, Temple; *Lester Karotkin*, Houston; *Everett P. Veatch*, Pasadena; *William Rosenblatt*, Wichita Falls; and *George R. Hermann*, Galveston, were present for the American College of Physicians postgraduate course in cardiology at the National Institute in Mexico, D. F., recently.

Dr. Lewis William Fetzer, Dallas, has been invited to become a member of the research committee of the President's Conference on Industrial Safety to be held in Washington, D. C., soon.

Dr. J. Gordon Bryson is now serving as mayor of Bastrop, according to the Bastrop *Advertiser*.

Dr. Henry W. Poetter, Kerrville, has been appointed acting superintendent of the Kerrville State Sanatorium, reports the Kerrville *Sun*.

Dr. Will Rogers, El Paso, was paid tribute for his thirty-four years of service to the City-County Hospital at the monthly hospital party given at the nurses home in August, states the El Paso *Times*.

Mrs. Lavinia Bishop Baird, wife of the late *Dr. R. W. Baird*, Dallas, died July 28, informs the Dallas *News*.

Births

To *Dr. and Mrs. Grady Baskin*, Galveston, a son, Thomas Grady Baskin, Jr., on July 7.

To *Dr. and Mrs. Peter M. Marcuse*, Houston, a daughter, Yvonne Lee, on July 3.

SOCIETY NEWS

Cass-Marion Counties Society

July 14, 1948

Twenty-four members, wives, and guests were present for a fish supper held by the Cass-Marion Counties Medical Society on July 14 at the camp of Dr. and Mrs. W. S. Terry on Carter's Lake, with the doctors of Jefferson as hosts.

Jefferson County Society

August 11, 1948

National Blood Bank Program of the American Red Cross—Raymond Frank Barnes, St. Louis.

Raymond Frank Barnes, St. Louis, administrator of the American Red Cross midwestern area medical services, was the principal speaker at the meeting of Jefferson County Medical Society in Beaumont on August 11. He discussed the Red Cross blood bank program.

Lamar County Society

August 5, 1948

(Reported by Thomas E. Hunt, Jr., Secretary)

Functional Heart Murmurs—J. L. Jopling, Paris.

Lamar County Medical Society, meeting August 5 in Paris, heard a discussion of functional heart murmurs by J. L. Jopling, who illustrated his talk with recordings of heart murmurs. He pointed out that 50 per cent of systolic murmurs are of no importance, but that diastolic murmurs indicate organic heart disease. He emphasized the necessity of taking a careful history in diagnosing heart disease.

Lubbock-Crosby Counties Society

August 3, 1948

(Reported by Roy G. Loveless, Secretary)

Modern Concepts of Diabetic Management—Ivan G. Mayfield, Lubbock.

Twenty-eight members and guests attended the August 3 meeting of Lubbock-Crosby Counties Medical Society in Lubbock. The scientific program consisted of a discussion of diabetic management in which Ivan G. Mayfield stressed the importance of teaching diabetic patients to care for themselves.

Mr. J. Louis Neff, Houston, executive director of the Texas Division, American Cancer Society, spoke briefly of the cancer movement and offered the facilities of the cancer society in the establishment of a diagnostic cancer clinic in Lubbock if such a program meets with the approval of the county medical society.

H. H. Latson, Amarillo, district councilor, and E. A. Rowley, Amarillo, trustee of the State Medical Association, talked of district matters.

The president, R. C. Douglas, Lubbock, gave a report for the committee on public health in the absence of the chairman. The committee recommended (1) a full-time assistant health officer; (2) separation of public health from welfare service; (3) modernization of public health laws; (4) employment of a school physician by the Lubbock Independent School District; (5) erection of structures by the city and county to coordinate the cancer society, blood bank, city laboratories, tuberculosis society, and other similar health agencies. Motion was made by Allen T. Stewart, seconded by O. R. Hand, that the report be endorsed and that the committee continue study of the problems and introduce a full discussion at the next meeting of the society.

John L. Cobb, Slaton, and Ennis E. Moss, Lubbock, were elected to membership.

Pecos-Jeff Davis-Presidio-Brewster Counties Society

August 1, 1948

(Reported by W. E. Lockhart, Secretary)

Skin Lesions of Venereal Diseases—J. C. Kern, Sanderson.

Pecos-Jeff Davis-Presidio-Brewster Counties Medical Society met August 1 at the residence of John W. Pate, Sanderson. Four members and six visitors were present.

J. C. Kern presented 130 slides prepared at Johns Hopkins University showing skin lesions of venereal diseases.

Seventh District Society

July 29, 1948

(Reported by David Wade, Secretary)

The Seventh District Medical Society held a social meeting July 29 at the ranch of G. F. Thornhill, near Austin. Several political candidates spoke. Officers for the coming year were elected as follows: David Wade, Austin, president; Jay J. Johns, Taylor, vice-president; and Joe W. Bailey, Austin, secretary-treasurer. A midwinter clinical meeting will be held in Austin.

Twelfth District Society

July 13, 1948

(Reported by H. F. Connally, Jr., Secretary)

Shoulder-Hand Syndrome—C. Collum Smith, Waco.
Surgical Diseases of Pancreas—Edgar J. Poth, University of Texas Medical Branch, Galveston.

Sympathectomy in Peripheral Vascular Disease—Harvey Mac Richey, Waco.
Management of Diabetes—Raymond Gregory, University of Texas Medical Branch, Galveston.
Analysis of Tonsillectomy and Adenoidectomy—Ernest A. Johnson, Waco.
Injuries to Carpal Bones—Howard R. Dudgeon, Jr., Waco.
Diverticulosis of Duodenum—Bromley S. Freeman, Veterans Administration Hospital, Temple.

The Twelfth District Medical Society held its semi-annual meeting July 13 at Bryan. The scientific program outlined above was presented. At noon the Brazos-Robertson Counties Medical Society was host to members of the District Medical Society and Auxiliary for luncheon. Dr. Charles I. Spurr of the M. D. Anderson Foundation, Houston, was the luncheon speaker.

AUXILIARY NOTES

Officers of the Woman's Auxiliary to the State Medical Association of Texas: President, Mrs. S. M. Hill, Dallas; President-Elect, Mrs. Joseph B. Foster, Houston; First Vice-President (Organization), Mrs. O. W. Robinson, Paris; Second Vice-President (Physical Examination), Mrs. Robert F. Thompson, El Paso; Third Vice-President (Hygeia), Mrs. J. C. Terrell, Stephenville; Fourth Vice-President (Program), Mrs. Edward W. Coyle, San Antonio; Corresponding Secretary, Mrs. Cecil O. Patterson, Dallas; Recording Secretary, Mrs. R. E. Clark, Memphis; Treasurer, Mrs. V. M. Longmire, Temple; Publicity Secretary, Mrs. H. S. Renshaw, Fort Worth; Parliamentarian, Mrs. J. C. Sharp, Corpus Christi.

STATE EXECUTIVE BOARD TO MEET

The Executive Board of the Woman's Auxiliary to the State Medical Association will meet September 22 at 2 o'clock in the home of Mrs. Samuel M. Hill, Dallas, President. Plans for the autumn and winter will be discussed by officers and committees. Mrs. Hill will be hostess at dinner and entertainment during the evening.

DEATHS*

Dr. John Wyeth Ray, Houston, Texas, died August 20, 1948.

Born in Dillon, S. C., on February 14, 1908, Dr. Ray was the son of Archibald C. and Ida C. Ray. He attended high school at Pittsboro, N. C.; received his bachelor of arts degree from the University of North Carolina, Chapel Hill; and was graduated in medicine from the Louisiana State University Medical School, New Orleans, in 1937. He served an internship at the City Memorial Hospital, Winston-Salem, N. C., and was house surgeon at the Nan Travis Hospital, Jacksonville, Texas, before beginning private practice at Timpson in 1940. After serving for some months as a first lieutenant with the Army Medical Corps, Dr. Ray returned to practice in December, 1941, at Houston, where he was active until shortly before his death.

Since 1940 Dr. Ray had been a member of the State Medical Association and American Medical Association, first through Shelby-San Augustine-Sabine Counties Medical Society, and later through Harris County Medical Society. He was a member and deacon of the First Presbyterian Church and a member of the East End Optimist Club.

Dr. Ray is survived by his wife, the former Miss Grace Duke of New Orleans, whom he married in 1934; his mother, Mrs. Archibald C. Ray, Sr., Richmond, Va.; four sisters, Mrs. R. J. Saville, State College, Miss.; Mrs. Glenn Fry, Columbus, Ohio; and Mrs. J. L. Wall and Mrs. Horace G. Porter, Richmond, Va.; and two brothers, the Rev. Archie C. Ray, Hammond, La., and William L. Ray, Richmond, Va.

*An obituary ordinarily will not be published more than four months after date of death. Cooperation in reporting deaths of physicians and in furnishing appropriate biographical material promptly is solicited.

Dr. Marvin Arnold Gantt died at his home in Houston, Texas, on July 1, 1948, of carcinoma of the liver.

The son of the Rev. Albert G. and Emma Catherine (Browning) Gantt, Dr. Gantt was born November 22, 1877, in Summerville, N. C. He attended high school and did college academic work in North Carolina, and was graduated in medicine from the Louisville Medical College, Louisville, Ky., in 1904. After serving as an intern and resident for three years at the Norton Memorial Infirmary, Louisville, Dr. Gantt moved to Texas. He practiced for three years each at Dallas and San Benito before locating in 1914 at Houston, where he was active until his retirement in 1947.

Throughout his professional career in Texas, Dr. Gantt was a member of the State Medical Association and American Medical Association through the

DR. MARVIN A. GANTT

Dallas and then the Harris County Medical Societies. Shortly before his death he was nominated for honorary membership in the Harris County Society. He was a member of the South Texas District Medical Society and the Houston Academy of Medicine. Dr. Gantt was a charter member of the Houston Masonic Lodge and a member of Scottish Rite bodies, the Arabia Temple Shrine, and the Methodist Church.

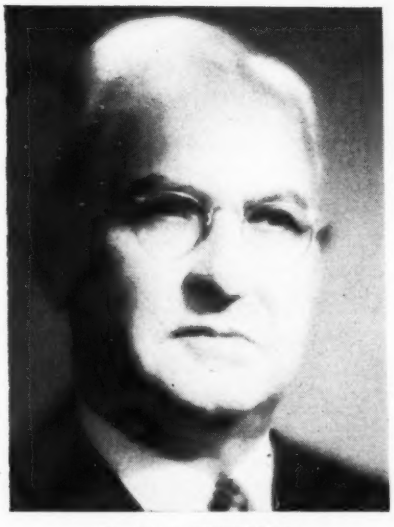
On June 29, 1929, in Houston, Dr. Gantt married Miss Minnie Ola Law, who survives. Also surviving are a son and a daughter by a previous marriage, Horace Gantt and Mrs. Marveen Schaele, both of Hollywood, Calif., and one brother, G. D. Gantt, Houston.

Dr. Herschel Frank Connally, former President of the State Medical Association, died July 16, 1948, at his home in Waco, Texas, of coronary occlusion.

Dr. Connally was born at Comanche Springs, near McGregor, on January 6, 1878, the son of Thomas G. and Mary Samanthia (Christian) Connally. His early education was received at McGregor, and his medical education at Vanderbilt University, Nashville, Tenn., from which he was graduated in 1900. He did postgraduate work at the New York Polyclinic, Chicago Post Graduate School, Mayo Clinic, Massachusetts General Hospital, and Johns Hopkins

University. He began practice in Bruceville, moved to Eddy in 1907, and then in 1912 to Waco, where he limited his practice to general surgery. During World War I, Dr. Connally served with the Army Medical Reserve Corps, first as major and then lieutenant colonel. He was chief of surgical service and then in command of Base Hospital 90 in France. While in France, Dr. Connally was given a post-graduate course in plastic surgery in the Hospital De-La Petie in Paris. Upon his discharge in 1919, Dr. Connally resumed his practice in Waco. He was the first chief of staff of the Hillcrest Memorial Hospital, which was opened in 1920; was consulting surgeon for the Veterans Administration Hospital from the time it was established in 1931; and was a member of the surgical staff of Hillcrest and Providence Hospitals. He was medical examiner of the Amicable Life Insurance Company and for many years local surgeon for the Missouri-Kansas-Texas Railroad.

Dr. Connally was a charter member of the McLennan County Medical Society, which was organized in 1903, and held continuous membership through that society in the State Medical Association and American Medical Association. He was also a member of the Twelfth District Medical Society. He was president of the McLennan County Medical Society, councilor of the Twelfth District from 1934 through 1942, chairman of the Board of Councilors, vice-president of the State Medical Association in 1918 and 1932; President-Elect in 1943, and President in 1944. He was chairman of the Section on Obstetrics and Gynecology in 1918 and of the Section on Surgery in 1937. Dr. Connally was appointed to the State Board of Medical Examiners in 1927 and was secretary of the board at the time of his death. He had been chairman of its committee on reciprocity



DR. H. F. CONNALLY

for a number of years and was on the board of directors of the Federation of State Medical Boards. He was a fellow of the American College of Surgeons.

The week before his death, Dr. Connally was honored by the Rotary Club of Waco for his perfect record of attendance for twenty-five years. In addition to being a member of the Rotary Club, Dr. Connally was a member of the Baptist Church, Masonic Lodge, and American Legion. During World War II he served on the local Draft Appeal Board. He was actively interested in politics, having been a city commissioner in 1925 and mayor of Waco in 1926. He was a political adviser to his cousin, Senator Tom Connally.

Preceding Dr. Connally in death in August, 1947, was his wife, the former Miss Clara Blailock, whom he married in 1905 in McGregor. He is survived by one son, Dr. H. F. Connally, Jr., Waco; two daughters, Mrs. Betsy Maness, Waco, and Mrs. Taylor Frazier, Bluefield, W. Va.; two brothers, Clarence Connally, Waco, and Tom Connally, Clarendon; and eight grandchildren.

Dr. Charles Russell Hartsook died at his home in Wichita Falls, Texas, on July 14, 1948, after a long illness.

Dr. Hartsook was born in Castlewood, Russell County, Va., in 1873. He attended Lebanon College in Virginia and the Medical College of Virginia, Richmond. He practiced for a short time in Natural Bridge, Va., before moving to Tulia, Texas, and then in 1911 to Wichita Falls.

Continuously since 1909 Dr. Hartsook had been a member of the State Medical Association and American Medical Association, first through Swisher County Medical Society and then through Wichita County Medical Society. He was councilor of the

DR. CHARLES R. HARTSOOK

Third District from 1916 until 1919 and a former president of the Wichita County Society. He was a former chairman of the board of directors of Wichita General Hospital, a fellow of the American College of Surgeons, and an organizer of the Texas Society of Ophthalmology and Otolaryngology. He was a member of the Methodist Church, the Masonic Order, Knights Templar, and Shrine. He was a charter member and past president of the Wichita Falls Rotary Club. He was an enthusiastic hunter, fisherman, and photographer of the out-of-doors, and was active in civic projects.

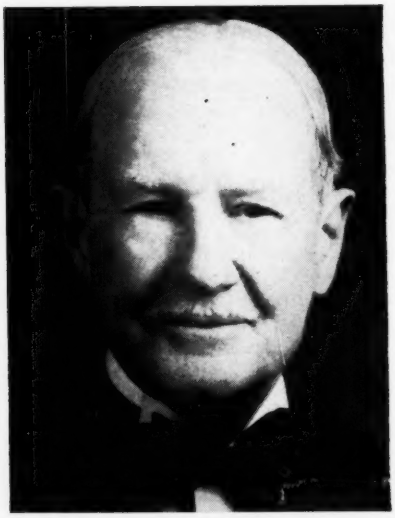
Surviving Dr. Hartsook is his wife, the former Miss Evaline Taylor of Richmond, Va.

Dr. Joseph A. Mullen, Houston, Texas, died June 8, 1948, at a local hospital.

The son of Edward and Sarah Mullen, Dr. Mullen was born August 18, 1866, in Philadelphia. Supposedly suffering from pernicious anemia, he spent

most of his early youth traveling in Europe and South America. Restored to health, he studied for two years at the Philadelphia College of Pharmacy and then was graduated in 1893 from the Jefferson Medical College, Philadelphia. The following year he went to Houston and became city and county oculist and aurist. For a number of years he was oculist and aurist for the Southern Pacific and Aransas Pass Railroads. He became associated with St. Joseph's Infirmary and was an honorary member of the staff at the time of his death. He and other associates organized at St. Joseph's Infirmary the first training school for nurses in Houston. Dr. Mullen retired from active practice in 1928.

Throughout his professional career Dr. Mullen was a member of the Harris County Medical Society, State Medical Association, and American Medical Association. Since 1934 he had been an honorary member of the State Medical Association. He served as secretary of the Section on Eye, Ear, Nose, and Throat in 1909. Dr. Mullen had been a member of the Southwestern Medical Association; Otolaryngological Society, Anglo-European; French Society of Ophthalmology; Ninth Congress of Ophthalmology;



DR. JOSEPH A. MULLEN

American College of Surgeons; and Sky Top Club, Sky Top, Pa. His hobby was the study of archeology. Dr. Mullen had been a member of Christ Episcopal Church since 1895 and had been a member of its vestry.

In Houston Dr. Mullen was probably best known as a patron of the fine arts, especially music and art. He organized the Houston Symphony Orchestra in 1931 and was its president from 1931 until 1934, during that time encouraging the support of prominent business men. He was chairman of the board of trustees of the Museum of Fine Arts at the time the museum building was constructed. Following the death of his wife, who shared his interest in the fine arts, Dr. Mullen and his son created a memorial fund for the establishment of an art library in the museum. They also provided funds for a medical library at St. Joseph's Infirmary.

Dr. Mullen married Mrs. Ida Kirkland Tyler in 1898. The couple traveled extensively in Europe and Mexico prior to Mrs. Mullen's death January 8, 1944. They are survived by a son, Joseph Mullen, New York City.

Dr. Vincent Sylvester Pawelek, Jr., Houston, Texas, was killed August 6, 1948, in an automobile accident near Eunice, La. He and his wife, who was injured in the accident, were en route to New Orleans for a vacation.

Born November 5, 1918, in Falls City, Texas, Dr. Pawelek was the son of Vincent S. and Eleanor Pawelek. He was graduated from the Karnes City High School and from St. Mary's University, San Antonio, from which he received a bachelor of science degree in 1939. His degree of doctor of medicine was obtained from Baylor University College of Medicine, Dallas, in 1943. He served an internship at the Robert B. Green Memorial Hospital, San Antonio, and then for two years was on duty with the Army, first with the Medical Administrative Corps Reserve as a lieutenant and then with the Transport Command in the Atlantic Theater as a captain. Upon his discharge in 1946, Dr. Pawelek became associated with his uncles, Drs. I. L. and Louis G. Pawelek, in Houston, where he was in practice at the time of his death.

Since 1944 Dr. Pawelek had been a member of the State Medical Association and American Medical Association, first through Bexar County Medical Society and later through Harris County Medical

DR. VINCENT S. PAWELEK, JR.

Society. He was also a member of the South Texas District Medical Society. He was a member of the Catholic Church, Knights of Columbus, Catholic War Veterans, Polish National Alliance, and Ex-Students Association of St. Mary's University.

Dr. Pawelek married Miss Hazel Patricia Lenore Green in Tulsa, Okla., on January 5, 1946. He is survived by his wife; an infant son, William Louis Pawelek; his parents, Mr. and Mrs. Vincent S. Pawelek, Falls City; one sister, Mrs. Franklin Ruhnke, Mission; and one brother, Albert Francis Pawelek, Falls City.

CORRECTION

Dr. Emanuel Toomim. — The surname of Dr. Emanuel Toomim, Beaumont, Texas, who died April 15, 1948, inadvertently appeared as "Toomin"" in the obituary published in the August, 1948, JOURNAL (page 342).